

Understanding Criminal Behaviour in Fetal Alcohol Spectrum Disorders: Neurocognitive
Deficits and Social Factors

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ABSTRACT

Individuals with fetal alcohol spectrum disorder (FASD) are more likely to be involved in the criminal justice system than individuals without FASD. Research shows that individuals with FASD are unable to learn from standard methods of punishment, such as incarceration. The objective of this research was to determine how young offenders with an FASD diagnosis differ from young offenders without a diagnosis in order to inform sentencing and treatment options for FASD offenders. Using a pre-existing database of court-referred young offenders, the data of 197 youths were compared. Eighty-six young offenders in this sample were diagnosed with FASD. Information was available on a number of neurocognitive variables, such as cognition, memory, attention, achievement, and language, as well as social data, such as substance use, assistance in school, home stability, and criminal charges. Profile analysis was run on the neurocognitive data for young offenders with and without FASD. The social data were analyzed using a combination of correlation and one-way ANOVAs. Young offenders with FASD differed from young offenders without FASD on severity of impairment on the neurocognitive measures, with individuals with FASD scoring lower than the comparison group. There was no difference in the profile of neurocognitive deficiency between the groups, suggesting that young offenders with FASD have the same profile of impairments as other young offenders but to a more severe degree. There were not found to be any strong or moderate associations between the types of charges accrued and any neurocognitive measure, indicating that deficits likely do not directly lead to offending. Home stability between birth and age seven was particularly important as a protective factor for future crime, and having ever been in foster care was strongly related to number of charges. Current substance use of all kinds was associated with a higher number of charges. Youth with FASD are likely more at risk for criminal behaviour due to lower overall neurocognitive functioning, poor environmental stability, and an interaction of the two. Programs for people with FASD will be required throughout the life span and current correctional programs have yet to be developed for offenders with FASD.

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DEDICATION

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LIST OF ABBREVIATIONS

ABA	American Bar Association
ABAS	<i>Adaptive Behaviour Assessment System</i>
ADHD	Attention Deficit/Hyperactivity Disorder
APT	<i>Attentional Process Test</i>
ARBD	Alcohol-Related Birth Defects
ARND	Alcohol-Related Neurodevelopmental Disorder
AUDIT	<i>Alcohol Use Disorders Identification Test</i>
BERS-SF	<i>Behaviour and Emotional Rating Scale – School Functioning</i>
BFLT	<i>Biber Figure Learning Test</i>
BPI	<i>Behaviour Problem Index</i>
BRIEF	<i>Behaviour Rating Inventory of Executive Function</i>
CANTAB	<i>Cambridge Neuropsychological Tests Automated Battery</i>
CBA	Canadian Bar Association
CBC	Canadian Broadcasting Corporation
CBCL	<i>Child Behaviour Checklist</i>
CD	Conduct Disorder
CDC	Centre for Disease Control
CHIP-AE	<i>Child Health and Illness Profile – Adolescent Edition</i>
CMS	<i>Children’s Memory Scale</i>
CNS	Central Nervous System
COWAT	<i>Controlled One Word Association Test</i>
CPT	<i>Continuous Performance Task</i>
CRS	<i>Connor’s Rating Scale</i>
CSC	Correctional Service of Canada
CTT	<i>Consonant Trigrams Task</i>
CVLT	<i>California Verbal Learning Test</i>
D-KEFS	<i>Delis-Kaplan Executive Function Scale</i>
DSE	Disrupted School Experience
DSM-IV	<i>Diagnostic and Statistical Manual, Fourth Edition</i>

EEG	Electroencephalography
EVT	Expressive Vocabulary Test
FAE	Fetal Alcohol Effects
FAEE	Fatty Acid Ethyl Esters
FAS	Fetal Alcohol Syndrome
FASD	Fetal Alcohol Spectrum Disorder
fMRI	Functional Magnetic Resonance Imaging
FSIQ	Full Scale IQ
HBHC	Healthy Babies Healthy Children
ICCFASD	Interagency Coordinating Committee on FASD
IGT	<i>Iowa Gambling Task</i>
IOM	Institute of Medicine
IQ	Intelligence Quotient
IUFD	Intrauterine Fetal Death
KTEA	<i>Kaufman Test of Educational Achievement</i>
Leiter	<i>Leiter International Performance Scale</i>
LCT	<i>Letter Cancellation Test</i>
MAYSI	<i>Massachusetts Youth Screening Instrument</i>
MNTAP	<i>Minnesota Test of Affective Processing</i>
MOSS	<i>Manifestation of Symptomatology Scale</i>
MRI	Magnetic Resonance Imaging
NGRI	Not Guilty by Reason of Insanity
NLSY-79	National Longitudinal Survey of Youth
NEPSY	<i>Developmental Neuropsychological Assessment</i>
NES	<i>Neurobehavioural Evaluation System</i>
NVSRT	<i>Nonverbal Selective Reminding Memory Test</i>
OCTC	<i>Object Classification Task for Children</i>
ODD	Oppositional Defiant Disorder
PAE	Prenatal Alcohol Exposure
PCAP	Parent-Child Assistance Program
pFAS	Partial Fetal Alcohol Syndrome

PIQ	Performance IQ
PPVT	<i>Peabody Picture Vocabulary Test</i>
RCMP	Royal Canadian Mounted Police
ROC	Receiver Operating Characteristic
ROCF	<i>Rey-Osterrieth Complex Figure Test</i>
SIB	<i>Scales of Independent Behaviour</i>
SPSS	Statistical Product and Service Solutions
SRM-SF	<i>Sociomoral Reflection Measure – Short Form</i>
SSP	<i>Short Sensory Profile</i>
SSRS	<i>Social Skills Rating System</i>
TMT	<i>Trail Making Test</i>
TOPS	<i>Test of Problem Solving</i>
TRF	<i>Teacher Report Form</i>
VABS	<i>Vineland Adaptive Behaviour Scales</i>
VIQ	Verbal IQ
VSRMT	<i>Verbal Selective Reminding Memory Test</i>
WAIS	Wechsler Adult Intelligence Scale
WASI	<i>Wechsler Abbreviated Scale of Intelligence</i>
WCST	<i>Wisconsin Card Sorting Test</i>
WIAT	<i>Wechsler Individual Achievement Test</i>
WISC	<i>Wechsler Intelligence Scale for Children</i>
WJ	<i>Woodcock Johnson</i>
WMS	<i>Wechsler Memory Scale</i>
WMTB-C	<i>Working Memory Test Battery for Children</i>
WPPSI	<i>Wechsler Preschool and Primary Scale of Intelligence</i>
WRAML	<i>Wide Range Assessment of Memory and Learning</i>
WRAT	<i>Wide Range Achievement Test</i>

1. INTRODUCTION

Fetal Alcohol Syndrome (FAS) was first identified by Jones and Smith (1973) as a condition affecting the children of some women who consumed alcohol while pregnant. It is characterized by growth retardation, distinctive facial features, and central nervous system (CNS) deficits, including impairments in memory, attention, and cognition. While the diagnostic features for FAS have remained largely the same since its inception (Chudley, Conry, Cook, Looock, Rosales et al., 2005), researchers and clinicians have discovered that significant cognitive, emotional, and behavioural problems can exist in children who were exposed to alcohol in utero, but who do not demonstrate the full physical abnormalities of the disorder (Autti-Ramo, 2000; Fryer, McGee, Matt, Riley, & Mattson, 2007; Mattson & Riley, 1997; Streissguth & O'Malley, 2001). In 1996, the Institute of Medicine (IOM) proposed a diagnostic scheme for FAS involving five diagnostic categories:

- FAS with confirmed maternal alcohol exposure,
- FAS without confirmed maternal alcohol exposure,
- Partial FAS (pFAS) with confirmed maternal alcohol exposure,
- alcohol-related birth defects (ARBD), and
- alcohol-related neurodevelopmental disorder (ARND) (Institute of Medicine, 1996).

The term fetal alcohol effects (FAE) was originally used to describe those affected by alcohol but without full-blown FAS; however, it is believed to have resulted in an overdiagnosis of the role of alcohol in behavioural and cognitive problems (May, Gossage, Brooke, Snell, Marias et al., 2004). As such, researchers now recommend that FAE not be used in any clinical capacity and that the IOM classification be used instead. In addition, ARBD is seen as more of a series of congenital abnormalities, and can be present without any evidence of CNS dysfunction (Chudley et al., 2005); as such, it is recommended that ARBD no longer be considered as falling under the fetal alcohol spectrum.

1.1. Diagnostic Considerations

Currently there are numerous and varied problems with accurate diagnosis of FAS and other FASD. The disorders are complex, and present with a broadly inconsistent phenotype (Aase, 1994; Burd, Martsolf, Klug, & Kerbeshian, 2003; Chudley et al., 2005). Individuals exposed prenatally to alcohol can range from those with severe growth restriction and

intellectual disability to normal growth and average intellectual functioning (Chudley et al., 2005). Even amongst those without the physical morphology, there are lifelong and irreparable impairments in other areas of cognitive functioning (Chudley et al., 2005; Conry & Fast, 2000; Mattson & Riley, 2000; Roebuck, Mattson, & Riley, 1999). In fact, it is argued that the majority of people with FASD do not demonstrate growth deficits and have few, if any, of the characteristic facial dysmorphic features (Chudley Kilgour, Cranston, & Edwards, 2007; May et al., 2004). These forms of FASD that are considered less severe, while more common, are also more difficult to diagnose. Despite their best efforts, clinicians have been unable to determine a behavioural and/or cognitive profile of deficits unique to individuals affected by prenatal alcohol exposure (PAE; Caprara, Nash, Greenbaum, Rovet, & Koren, 2007; Chudley et al., 2005; Nash, Stevens, Rovet, Fantus, Nulman, et al., 2013). The diagnostic criteria are, in general and with the exception of the characteristic facial features, relatively common disabilities seen in a range of other disorders, and are sporadically present in normal individuals as well (Aase, 1994). Even the distinctive FAS facial features can be similar (but not identical) to what is found in children exposed to other embryotoxic agents, such as anti-convulsants and maternal PKU syndrome (Waisbren, Hanley, Levy, Shifrin, Allred et al., 2000), and such children often display similar growth deficiencies and developmental and behavioural disabilities (Chudley et al., 2005).

Due to the variability in presentation and overlap with other disorders, misdiagnosis and comorbidity are of serious concern. Commonly, people with FASD who have a comorbid mental disorder are not recognized as having FASD (Substance Abuse and Mental Health Service Administration, 2006). Research has found high rates of attention deficit/hyperactivity disorder (ADHD), depression, oppositional defiant disorder (ODD), and conduct disorder (CD) in individuals with heavy prenatal exposure to alcohol (Fryer et al., 2007), with ADHD being the most reported co-occurring mental disorder (Doig, McLennan, & Gibbard, 2008). Nanson and Hisock (1990) determined that ADHD was present in as many as 70% of children with FASD in their sample. In other studies, children with FASD have been found to be twice as likely to have a previous diagnosis of ADHD (Nash et al., 2013). In addition, Steinhausen, Willms, and Spohr (1993) found a strong association exists between the severity of the morphological damage and psychopathology. This association is present even in school-aged children despite the decrease in facial morphology over time. When facial features are not present, children with FASD are likely to be misdiagnosed with ADHD, conduct disorder (CD), oppositional defiance disorder (ODD),

intermittent explosive disorder, and major depressive disorder (Dubovsky, 2008). Misdiagnosis can lead to the mothers of labeled children being stigmatized by a society that now recognizes the dangers of prenatal alcohol exposure and casts harsh judgment on women who drink while pregnant (Aase, 1994; May et al., 2004). It is likely that an individual with FASD will exhibit the aforementioned emotional and behavioural problems and meet criteria for a formal diagnosis of ADHD, CD, etc; however, to miss the FASD diagnosis is to misunderstand the etiology of his/her impairments. Once a diagnosis is reached, the search for other potential causes is typically abandoned. As a result, interventions are likely to be tailored to address only part of the issue and therefore will potentially be inappropriate and ineffective (Astley, 2006).

Further compounding the difficulty in FASD diagnosis, most criteria require a reported history of maternal drinking during pregnancy, particularly in the absence of the characteristic facial dysmorphology (Astley, 2006; Burd et al., 2003; Caprara et al., 2007; Chudley et al. 2005; IOM, 1996). However, this information is often very difficult to accurately obtain (Astley 2006; Burd et al., 2003; Burd, Cotsonas-Hassler, Martsolf, & Kerbeshian, 2003). Birth and medical records are commonly unavailable or incomplete (Chudley et al., 2005, 2007; May et al., 2004), particularly for children in foster or adoptive settings. This is even more of a problem with children who have been adopted from another country. Women may be reluctant to admit to drinking while pregnant, or have difficulty correctly recalling the frequency and amount of alcohol consumption, especially in retrospective studies (Astley, 2006; Chudley et al., 2005). Commonly, the child is no longer with the birth mother, and so a reliable collection of prenatal exposure is not possible (Astley, 2006; Conry & Fast, 2000). Due to the challenges of collecting valid prenatal exposure information, some diagnostic systems have relaxed their standards so that a diagnosis of FAS alone can be made without a confirmation of maternal drinking, based on the presence of the distinctive facial characteristics (Chudley et al., 2007; IOM, 1996); certain criteria even permit for a diagnosis of pFAS without confirmed exposure (May et al., 2004).

Past research in the field of FASD has suffered from a number of methodological concerns in regard to gathering information about alcohol consumption. For instance, different projects have different definitions of what constitutes “light”, “moderate”, and “heavy” drinking (Moyer, 2013). Additionally, the drinks one reports may be averaged together, which could lump together women with drastically divergent drinking patterns and obscure binge drinking. Binge drinking is increasingly looking to be more related to FASD than infrequent, small doses (Fraser,

Muckle, Abdous, Jacobson & Jacobson, 2012; Hepper, Dorman, & Lynch, 2012; Meyer-Leu, Sakari, Daeppen, Derlaz, & Gerber, 2011).

Recognizing the serious implications of an inadequate prenatal exposure history, researchers are currently working to discover objective, biological methods of determining prenatal alcohol exposure, with hope that such procedures would simplify the diagnostic procedure. Biomarkers offer one such possibility (Caprera et al., 2007). Past research has determined there is evidence for an association between alcohol consumed while pregnant and maternal serum biochemical markers (Wurst, Alexson, Wolfersdorf, Bechtel, Forster et al., 2004). It is the hope of physicians and other health professionals that such biological indicators can offer an alternative to depending on the self-reported drinking habits of the mother.

Other research is focused on meconium fatty acid ethyl esters (FAEE) screening (Caprera et al., 2007). FAEE are compounds that only develop when an infant has been exposed to Ethyl alcohol, and can be measured in meconium (an infant's first stool) following birth (MacLeod & Koren, 2011). MacLeod and Koren recommend that the meconium of all infants should be routinely screened for the presence of FAEE metabolites. Given these recent developments, one can be optimistic that in the future, the question of prenatal exposure may be more easily answered.

One pilot study looked into an FAEE screening program in London, Ontario, to determine if women would engage willingly in such an endeavor (Zelner, Shor, Lynn, Roukema, Lum, et al., 2012). Every woman who delivered at St. Joseph's Health Care in London, Ontario between November 1, 2008 and May 31, 2010 was offered free meconium screening and follow up services; the authors do not note how many women took advantage of this offer. The researchers identified a site known to have high previous rates of FAEE-positive meconium through anonymous testing. Through this screening program, they detected one newborn exposed to heavy alcohol exposure in utero; this infant had average body weight, passed a hearing test, and registered satisfactory Apgar scores. The mother of this infant reported that she had had depression several years prior to the child's birth. She acknowledged smoking cigarettes, denied street drug use, and admitted to drinking alcohol daily before learning of her pregnancy. The obstetrics unit was able to refer this mother to the Healthy Babies Healthy Children (HBHC) program for support from a home nurse and periodic neuropsychological assessments of the child at no cost. It appears that in this case, an FAEE screening program was able to identify and

assist an at-risk mother with her child who may be negatively impacted by the use of alcohol during pregnancy. From the data presented, the cost effectiveness of the program cannot be known, as the number of screened participants is not provided but it appears to have been effective in this case. Unfortunately, this study was too recent to report any follow-up for the intervention.

Further contributing to diagnostic challenges regarding FASD, diagnosis in adulthood is more difficult than diagnosis in childhood. In fact, the ideal age to diagnose is between two and eleven years old (Chudley et al., 2007), despite the commonly held misconception amongst mental health professionals that the best time for diagnosis is at birth (Wedding, Kohout, Mengel, Ohlemiller, Ulione, et al., 2007). This is partly because the facial features appear to diminish or change with age (Aase, 1994; Conry & Fast, 2000; Streissguth, Aase, Clarren, Randels, LaDue, et al., 1991), and thus become less diagnostically useful. Some researchers argue that the morphological damage is not significantly altered with time (Chudley et al., 2007), although the evidence cited appears to be their own experience as opposed to systematic evaluation. There does not appear to be a clear consensus in the literature regarding the course of development of the facial features, nor their diagnostic utility in adults.

Perhaps the greatest impediment to diagnosing FASD is the lack of training clinicians and other professionals receive in identifying and managing FASD. An American survey of 1417 clinically active, doctoral-level American Psychological Association members revealed that 68% felt “very unprepared” or “somewhat unprepared” to identify FASD (Wedding et al., 2007). Only 4% felt “very prepared”. Underdiagnosis and underreporting are a problem because of a lack of training and expertise in the area of FASD (Chudley et al., 2005; Looock, Conry, & Cook, 2005). An international survey of FASD assessment clinics revealed vastly divergent and variable assessment procedures, with little standardization in procedure, testing, and diagnostic criteria used (Peadon, Fremantle, Bower, & Elliott, 2008). This can lead to individuals with the same symptoms receiving a wide variety of different diagnoses (Burd et al., 2003). A lack of diagnostic training was identified by 71% of clinicians as the most common barrier to diagnosing FASD (Wedding et al., 2007). The problem is even more pronounced in rural communities, which often lack the professionals qualified to make a diagnosis (Chudley et al., 2005). Travelling to urban centres to receive a proper assessment can be very expensive in terms of time and resources for affected families. While telehealth strategies may offer a solution to problems

of distance, these methods still require a telehealth facility be in place with the necessary equipment. As of yet, telehealth centres in rural areas are uncommon (Gagnon, Duplantie, Fortin, & Landry, 2006), and the centres that exist can be affected by limited communication services, such as restricted Internet access (Nelson, 2009). Also relevant in the context of diagnosing FASD is that the majority of neuropsychological measures have yet to be validated to be administered remotely (Jacobsen, Sprenger, Andersson, & Krogstad, 2003).

1.1.1. FASD Diagnostic Criteria

There is no current international consensus amongst health professionals as to the requisite criteria for a diagnosis. The first criteria were those proposed by Jones and Smith (1973), which have not radically changed from their initial conceptualization. The ongoing dissent among professionals appears to be how best to measure or determine each criterion, rather than whether such criteria are relevant. The fundamentals of diagnosis remain prenatal alcohol exposure, growth retardation, characteristic facial features, and central nervous system dysfunction. However, experts disagree on the extent of deficit required for impairment, how to measure such deficits, and the strictness applied to each criterion.

Following Jones and Smith (1973), the next attempt to clarify and determine accurate diagnosis was undertaken by the Institute of Medicine (IOM) in 1996. Under these guidelines, the diagnostic possibilities were expanded from just FAS to FAS, partial FAS, ARND, or ARBD; the criteria also allow for a diagnosis of FAS without confirmed prenatal alcohol exposure, as long as the facial features are present (Hoyme, May, Kalberg, Kodituwakku, Gossage et al., 2005). The IOM FASD criteria have been criticized for being vague, as the categories are not supplied with specific parameters for diagnosis (Hoyme et al., 2005; May et al., 2004). As well, there are not guidelines for the assessment of the “complex behavioural or cognitive difficulties” encountered by FASD affected individuals (May et al., 2004). So while a behavioural/cognitive phenotype is proposed, it is not specifically characterized. Additional criticisms of the IOM criteria include an inadequate collection of family history and poor clinical definition of the ARBD and ARND categories.

The 4-Digit Diagnostic Code (also known as the Washington Criteria) endeavored to improve upon the IOM guidelines by standardizing the diagnostic criteria. Developed in conjunction with the University of Washington FAS Diagnostic and Prevention Network of FASD clinical experts and using the extensive records of 1014 patients with known prenatal

exposure to alcohol (Astley & Clarren, 2000), it was first printed in 1997, and was updated in 1999 and 2004 (Astley, 2006). While the 4-Digit Code still addressed guidelines across the diagnostic spectrum, it also provided a strict criterion for defining “abnormal”: that is, two standard deviations at or below the mean. This is the standard medical cutoff for abnormality. Prior to the 4-Digit Code, diagnostic features were rated as either present or absent; the 4-Digit Code permitted features to be rated along a continuum (Astley, 2004). Each indication of FASD is rated on a four point likert scale, reflecting the magnitude of the expression for each feature, in the following order: 1) growth deficiency; 2) the FAS facial phenotype; 3) brain damage/dysfunction. This method of diagnosis has proven to be especially useful when judging the typical facial characteristics (Astley, 2006). However, the code has been criticized for being overly confusing and impractical for clinical practice use (Hoyme et al., 2005; Manning & Hoyme, 2007; May et al., 2004). Under this system, 256 total possible combinations are subsumed into 22 diagnostic categories. In addition, critics state that the 4-Digit code suffers from many of the same ambiguities as the IOM criteria. In fact, the risk for overdiagnosis using this system may be heightened, as any prenatally exposed child with a disability could be assigned a diagnostic classification, regardless of the cause for the impairment (May et al., 2004).

The next attempt to clarify the diagnostic criteria was produced by the Centre for Disease Control (CDC; 2002). It was hoped that the CDC guidelines would provide improved standardization and better correspond to guidelines employed in other centres in both America and internationally. The criteria were developed through a combination of research, clinical expertise and information from the families of individuals with FASD regarding their observations of the physical and neuropsychological features. The guidelines sought to update and refine past criteria. A strength of this system is that the facial dysmorphology is clearly defined, and incorporates age and racial norms. The criteria recognize that growth deficiencies may have resolved themselves, and so include any history of growth deficiency as part of the diagnostic criteria. As well, the criteria document added a list of available services for the impaired and their family. However, the CDC criteria suffer from many of the same faults of previous systems, as well as several new ones. Due to the lack of specificity of the CNS dysfunction in the literature, the guidelines do not provide strict criteria so much as general guidelines for determining CNS dysfunction (CDC, 2002). These guidelines were developed

based on the relatively small sample of 22 expert FASD clinicians and their personal experiences with the population. Clinicians were asked to identify the five CNS domains most vital to diagnosis, but fail to specify the basis of the five-domain limit. By these criteria, an impaired IQ is sufficient representation of CNS dysfunction to warrant diagnosis. The CDC criteria also use a diagnostic cutoff value of one standard deviation below the mean, or a score that is at or less than the tenth percentile (Astley, 2006). In standard clinical practice, these scores would still be considered in the normal range.

At approximately the same time as the CDC published their guidelines, two other standards for FASD diagnosis were released. The first was a revision of the IOM criteria by Hoyme and colleagues (2005), quite a significant departure from other systems. In some ways, these guidelines were more stringent; too stringent certain researchers have argued, as criteria for CNS dysfunction was restricted to structural abnormalities only (Astley, 2006). Commonly, the most disabling component of CNS dysfunction is the accompanying cognitive and behavioural impairment (Astley 2006; Chudley et al., 2007; Conry & Fast, 2000; Streissguth, Barr, Kogan, & Bookstein, 1997). According to Hoyme's system, FAS can be diagnosed if structural abnormalities are present, even if dysfunction is absent (Astley, 2006). In addition, under these guidelines, evidence of prenatal alcohol exposure is not enough; alcohol exposure is required to be excessive (Hoyme et al., 2005). Excessive alcohol use is defined as "substantial regular intake or heavy episodic drinking", but further clarification is not provided. This is contrary to the approach other models take, as there is currently no known exposure threshold under which all fetuses are safe from FAS (Arendt & Farkas, 2007; Astley, 2006; Sokol, Delaney-Black, & Nordstrom, 2003).

In other ways, the Hoyme guidelines are more liberal, as the criteria for small head circumference and facial features are relaxed. Unlike the 4-Digit Code, for which a label of deficiency required measurements be two standard deviations below the mean, Hoyme's recommendations considered head circumference deficient at or below the 10th percentile (Hoyme et al., 2005). Regarding the facial features, only two of the three generally agreed upon features need be present to receive a diagnosis of FAS. Research has determined that the Hoyme facial phenotype may be too relaxed. In one sample, it was found that 88.2 percent of participants who met criteria for the Hoyme FAS face did not have FAS, as determined by a multidisciplinary team using the 4-Digit Code (Astley, 2006). Perhaps most concerning are the

criticisms leveled against Hoyme's methodology. His team used a sample exclusively of South African and Native American children when developing the facial feature norms (May et al., 2004), which is not representative of a broader North American population. As well, they were forced to rely on the mother's self-reported drinking while pregnant (Hoyme et al., 2005). The problems with self-reports of drinking have been discussed above. Adding to the problem in this particular sample was that the adoption records of their American sample were sealed, which rendered them unable to obtain any information regarding prenatal alcohol exposure for many of their American participants (May et al., 2004). It seems difficult to generalize from this sample to a general North American public. While Hoyme and colleagues made efforts to clarify and specify diagnostic criteria, ultimately their goal of proposing a comprehensive and applicable guide for diagnosing FASD was not successful.

In Canada, Health Canada has developed national guidelines for diagnosing FASD that have been accepted and implemented across the country. Canada is the only nation to have nationally accepted standards for FASD diagnosis (Chudley et al., 2005). As these criteria have been widely adopted for use in Canada, it would serve to examine them in greater depth.

1.1.2. Fetal Alcohol Spectrum Disorder: Canadian Guidelines for Diagnosis

While there is no international consensus on FASD diagnostic guidelines, professionals in Canada have agreed to a set of criteria, standardizing diagnosis across the country. The Canadian guidelines for diagnosing FASD were developed through extensive, face-to-face interviews with experts in FASD from Canada and the United States (Chudley et al., 2005). Participants were both academic and clinical, coming from all levels of government and professional organizations. Recommendations and criteria were developed, and then returned to the participants for further feedback. From this feedback, the guidelines were agreed upon. The consensus generated by the expert panels proposed nine domains of CNS dysfunction to be examined in a neurobehavioural assessment: neurological signs, brain structure, cognition, communication (receptive and expressive), academic achievement, memory, executive functioning and abstract reasoning, attention, and adaptive behaviour and social skills (Chudley, 2005). A diagnosis of FASD, according to the criteria, would require impairment in three of the aforementioned domains.

The Canadian Diagnostic Guidelines (Chudley et al., 2005) improved upon previous guidelines in several ways, harmonizing the 4-Digit Code and IOM diagnostic criteria. As

previously mentioned, the guidelines stress a multidisciplinary team. Such teams are evidencing great success in assisting families with more consistency (Khan, Turner-Stokes, Ng, & Kilpatrick, 2008; McGonnell, Corkum, McKinnon, MacPherson, et al., 2009). Along a similar line, the guidelines advocate for the gathering of supplemental information from a variety of sources. The document discusses the importance of working with caregivers, a consideration rarely spared for them, despite their high rates of stress (Plant & Sanders, 2007; Tsai & Wang, 2009) and burnout (Gray-Stanley, 2009; White, Edwards, & Townsend-White, 2006). The guidelines suggest discussing in detail what a diagnosis of FASD would mean for them and their child. This can help to allay concerns and strengthen the relationships between the family/caregiver and the professional (Konrad, 2005; Winefield, Barlow, & Harvey, 1998). As previously mentioned, these guidelines are used by all the clinics in Saskatchewan and Canada that provide an FASD diagnosis, thus ensuring comparability of the diagnosis within the province.

Perhaps of most benefit is the specificity of the assessment. Chudley and colleagues (2005) stress the importance of considering the differences between domains, rather than merely looking for deficits. This is of particular assistance for cases in which the IQ and other cognitive domains are less impaired, as is often the case in individuals with ARND (Conry & Fast, 2000; Roebuck, Mattson, & Riley, 1999; Streissguth et al., 1991). Such attention to detail allows for interventions to be uniquely tailored to suit any individual's needs and strengths. Nor do the Canadian guidelines permit a diagnosis of FASD on the basis of any single indicator; the 4-Digit Diagnostic Code and the IOM model each allow for diagnosis on the basis of one criterion (Chudley et al., 2005).

People with FASD who go undiagnosed as such are at an increased risk for secondary disabilities (Chudley et al., 2007; Looch, Conry, & Cook, 2005). The disabilities encountered in an individual with FASD can be either primary or secondary to the organic brain damage (Buxton, 2004; Streissguth, Barr, Kogan, & Bookstein, 1997). Primary disabilities are those deficits that directly manifest the CNS dysfunction, whereas secondary deficits occur following birth, and could possibly be improved or avoided with proper care and adequate intervention. Some of the more common secondary disabilities are mental health problems, difficulties in school, substance abuse, inappropriate sexual behaviour and legal problems (Streissguth et al., 1997). Such problems have been found to be a drain on society's mental health, educational, and

correctional resources; estimates of resources spent in the United States annually as a result of FASD range from \$321 million (Streissguth et al., 1991) to \$4 billion (Lupton, Burd, & Harwood, 2004) to \$8.5 billion (Popova, Stade, Bekmuradov, Lange, & Rehm, 2011). In Canada, recent estimates for annual expenditures related to FASD range from \$344.2 million to \$5.3 billion, using conservative estimates of prevalence (Popova, et al., 2011). The majority of these resources are related to the secondary disabilities found in people with FASD (Peadon et al., 2008); however, costs associated with increased involvement with the justice system were not factored into any previous cost estimates (Popova et al., 2011). Since the development of the Canadian Guidelines, much research has examined the accuracy of these criteria. It is useful to see how the diagnostic criteria fare when subjected to objective evaluation.

1.1.3. FASD and CNS Dysfunction.

1.1.3.1. Neurological signs. Individuals who were prenatally exposed to alcohol evidence clinically impaired development in motor skills. Kalberg, Provost, Tollison, Tabachnick, Robinson, and colleagues (2006), looked at the motor development of children with FAS, when compared with children with a history of prenatal alcohol exposure but not FAS, as well as a control group of children with no record of PAE. The children were all between the ages of 20 and 68 months. Using the *Vineland Adaptive Behaviour Scales (VABS)*, children with FAS were found to exhibit clinically significant delays in the development of their motor skills. In addition, fine motor skills were found to be more impaired than gross motor skills in the FAS sample.

People with FASD typically evidence problems with ocularmotor control (Green, Mihic, Brien, Armstrong, Nikkel, et al., 2009). Green and colleagues examined impairments in sensory, motor, and cognitive processing in 89 youth with an FASD, aged between five and fifteen years old, when compared with a control group of 92 unexposed youths. Using saccadic eye movement testing, they determined that participants with an FASD had longer reaction times to the stimuli and more intra-subject variability in reaction times. In addition, the participants with FASD made more errors than controls. Individuals with ARND made significantly fewer errors than those with FAS or pFAS; however, all FASD-diagnosed participants were significantly different than controls.

Additional deficits have been found in eyeblink conditioning. In a sample of 98 children aged five, participants with FAS were unable to develop a conditioned response eyeblink in reaction to a puff of air (Jacobson, Stanton, Molteno, Burden, Fuller, et al., 2008). This is in

comparison to a non-PAE exposed control group, in which 75% of participants were able to develop the conditioned response. These results remain significant after controlling for IQ scores as well.

Sensory processing has been identified as one area of concern in those with FASD. In a retrospective study using the Washington State FAS DPN Clinical database, Franklin, Deitz, Jirikowic, and Astley (2008) looked to determine the characteristic and problematic behaviours of sensory-processing in children with FASD, aged five to ten. Franklin and colleagues examined the stored information on the *Short Sensory Profile (SSP)*, helpful in identifying sensory processing impairments, and the *Child Behaviour Checklist (CBCL)*, which is used to measure problem behaviours. A significant proportion of the sample displayed impairments in sensory processing and problem behaviours; unfortunately, the study did not include a non-PAE control group, and so conclusions regarding the severity of the deficits compared to non-exposed individuals cannot be drawn. Franklin and colleagues did find a significant relationship between sensory processing deficits and problem behaviours, and suggest that the deficits found in sensory processing in children with FASD may impact their ability to adaptively respond to their environment.

Differences have also been detected in auditory processing (Stephen, Kodituwakku, Kodituwakku, Romero, Peters, et al., 2012). Using a sample of 12 children diagnosed with FASD, compared to 19 healthy control peers, it was determined that those children with FASD were auditorially delayed compared to controls, even though their hearing was normal in general. The authors conclude from their findings that individuals with FASD likely suffer a significant central auditory delay without documented peripheral hearing loss.

Neural differences may also extend to areas of the brain known to be involved in inhibition. O'Brien, Norman, Fryer, Tapert, Paulus, et al. (2012) used fMRI to examine differences in neuronal activity between 20 heavy alcohol-exposed and 15 control participants; the sample consistent of children and youth from the age of eight to 18. Participants were asked to engage in a go/no-go task. The results indicated that youth who were exposed to alcohol demonstrated more activation in the left precuneus, cingulate gyrus, anterior cingulate, and right medial frontal gyrus during no-go trials (when they were being asked to inhibit their impulses), relative to the go-trials. Compared to the control group, the group of alcohol exposed participants

displayed greater frontal and parietal activation during attempts to inhibit responses, although both groups had similar rates of commission errors.

1.1.3.2. Brain structure. Research has shown that individuals with FASD demonstrate a reduction in overall brain volume. Coles, Goldstein, Lynch, Chen, Kable, and colleagues (2011) used structural MRI to calculate brain volume for regions of interest in 92 young adults. They compared three groups: one with participants with an ARND diagnoses, one for participants with FAS or pFAS (some form of facial dysmorphology), and control participants, with no prenatal exposure to alcohol. Coles and colleagues determined that individuals exposed to alcohol prenatally had significantly lower overall brain volume when compared to controls. In addition, the hippocampus in alcohol-exposed individuals was also found to be lower in volume than for controls. This last finding supports the research of Astley, Aylward, Olson, Kerns, Brooks, and colleagues in 2009. Astley and colleagues used magnetic imaging (MRI) and functional MRI (fMRI) to determine the presence of global and/or focal abnormalities in PAE individuals. They found a reduction in overall brain volume, with volume decreasing progressively across the diagnostic spectrum (as one moves from ARND to FAS). In addition, they discovered reduced volume in the frontal lobe when compared to non-PAE participants, as well as lower absolute volume of the caudate, the putamen, and the hippocampus.

Grey matter reduction appears to be a problem in individuals with FASD. Nardelli, Lebel, Rasmussen, Andrew, and Beaulieu (2011) examined 28 participants with FASD and 56 unexposed control participants for differences in six grey matter structures: hippocampus, amygdala, thalamus, caudate, putamen, and globus pallidus. Differences were detected using a structural MRI procedure. They determined that overall, the individuals with FASD displayed approximately 8% reduction for intracranial, white matter, and grey matter. However, the grey matter was disproportionately affected in the FASD group, as all six grey matter regions were reduced in comparison to controls. In this sample, grey matter was reduced between 10 and 19%; this was a greater reduction than was observed for intracranial volume, white matter, or cortical grey matter. The areas most affected were the basal ganglia substructures, specifically the globus pallidus and caudate. The authors also examined the subdiagnoses of FASD for differences but found no differences in brain volume between those with FAS, pFAS, and ARND, although their sample of FASD participants may have been too small to detect differences.

Interestingly, other research has also shown that white matter is disproportionately reduced in comparison to gray matter in prenatally exposed participants. In a study of 24 children diagnosed with FASD compared to 95 control children, Lebel, Rasmussen, Wyper, Walker, Andrew and colleagues (2008) used MRI diffusion tractography to examine white matter. In addition to overall brain volume (which this study also found to be significantly reduced), individuals with FASD displayed a greater reduction in white matter than gray matter when compared to controls, although both white and gray matter were significantly reduced.

In addition, abnormalities of the corpus callosum are consistent among those with FASD (Astley et al., 2009). In a study using MRI technology, Woznial, Mueller, Chang, Muetzel, Caros, and Lim (2006) compared structural brain abnormalities in 14 PAE children (ages ten to thirteen) to 13 matched controls. Those with a full diagnosis of FAS were excluded, as the researchers were interested in determining differences in less affected participants. Regardless, they found that even amongst the more mild forms of FASD, there were abnormalities of the corpus callosum, with the isthmus displaying a microstructural abnormality that suggests the tissue in this region is altered in PAE individuals. D'Anguilli, Grunau, Maggi, and Herdman (2006) reviewed the literature on studies using electroencephalography (EEG) to examine individuals with known PAE. They concluded that people with FASD evidence critical impairments in areas of the brain that allow for communication between different regions, specifically malfunctioning in areas such as the parietal and temporal lobes, which partially account for the integration of information received through the senses. These abnormalities could help account for the difficulty people with FASD have in encoding and recalling information, leading to impairment in their ability to learn from consequences.

It has been hypothesized that people with FASD may evidence a different developmental pathway when it comes to cortical thinning as well, with children with FASD showing an absence of cortical thinning typically seen in healthy development. In a sample of 21 participants with FASD aged eight to twenty-two, children and youth with FASD demonstrated thicker cortices than healthy controls in the bilateral inferior parietal, bilateral temporal, and right frontal regions (Sowell, Mattson, Kan, Thompson, Riley, et al., 2008). However, this finding has not been replicated in the literature. Zhou, Lebel, Lepage, Rasmussen, Evans, et al. (2011) examined regional variation in cortical thickness for 33 participants with FASD; the FASD participants were matched on age, sex, and handedness to a control group. The sample ranged from age six to

thirty. The authors assessed for cortical thickness, as well as the correlations between thickness and cognitive functioning in participants with FASD. They observed widespread cortical thinning in superior and middle frontal, superior parietal, inferior temporal, and occipital regions. They found no relationship between cognitive performance and cortical thinning overall; however, deficits related to executive functioning and inhibition, expressive and receptive language, learning and language functioning, motor skills, and visuospatial ability may be associated with thinning in the inferior and middle frontal, middle and inferior temporal, parahippocampal, motor, premotor, supplementary motor area, and occipital cortex. More research is required to fully elucidate the relationship between FASD, cortical thinning, and neurocognitive deficits.

1.1.3.3. Cognition. People with FASD consistently demonstrate lower full-scale IQ scores than control groups. In a sample of 78 children with an FASD diagnosis, Chasnoff, Wells, Telford, Schmidt, and Messer (2010), compared the participants on a number of neurodevelopmental functioning categories, including ability. Using scores on the *Wechsler Intelligence Scale for Children, Third Edition (WISC-III)*, Chasnoff and colleagues determined that participants with FAS scored significantly lower on the *WISC-III* when compared with participants diagnosed with pFAS or ARND; the pFAS and ARND groups did not differ significantly from each other. There are several limitations to this study, however; Chasnoff and colleagues did not employ a control group, and all the participants were either in foster care or adopted, restricting the generalizability of such findings. Still, given the norms of the *WISC-III*, conclusions as to the cognitive impairment of individuals with FASD can be made, as scores can be compared to age appropriate norms to determine deficits.

Howell, Lynch, Platzman, Smith, and Coles (2006) also examined intellectual ability in individuals with PAE. Using a longitudinal cohort, 265 participants of low socioeconomic status were evaluated with the *WISC-III*. Three groups were identified: 128 prenatally exposed to alcohol, 53 control students, and 84 students with special education placements. They found that students who were prenatally exposed produced significantly lower IQ scores than all other groups, across all domains with the exception of processing speed, on which the special education group also scored lower than the control group. Specific subtests that PAE individuals scored significantly lower on include Picture Completion, Coding, Block Design, Object

Assembly, Vocabulary, Arithmetic, Comprehension, and Symbol Search; the special education group evidenced more problems with Coding than the prenatally exposed group.

Using a sample of 170 children with PAE referred to a Motherrisk FASD clinic for academic, behavioural, or social problems, Nash et al. (2013) assessed for differences on a number of neurocognitive domains between children who received an FASD diagnosis and those who did not. 109 children were identified as having FASD. Like the present research, the control group, while not meeting criteria for a diagnosis, still evidenced behavioural difficulties warranting intervention, yet differences were still found between groups. Participants with FASD scored lower on FSIQ, Similarities, Vocabulary, Comprehension, Information, and Arithmetic.

This link between FASD and lower ability is repeatedly established in the literature (McGee, Bjorkquist, Riley, & Mattson, 2009). However, a diagnosis of FASD does not ensure an impaired IQ; only 24% of people with FAS and 7% of people with ARND have an IQ below the cut-off for mental retardation; that is, an IQ less than 70 (Streissguth et al., 2004). Often the deficits are varied. For example, Rasmussen, Horne, and Witol (2006) looked at 50 Canadian children diagnosed with an FASD in a clinically referred sample, again using the *WISC-III*, as well as the *Wechsler Preschool and Primary Scale of Intelligence, Revised (WPPSI-R)* for younger participants (participants ranged in age from six to fifteen, with an average age of nine years old). They found the average Full Scale IQ of their sample fell in the Below Average range (i.e., IQ's of 80-89), with significantly higher mean Performance IQ (PIQ) scores than Verbal IQ (VIQ); 41% of participants recorded a clinically significant Performance-Verbal split with PIQ being higher. The greatest deficits were seen in areas of Verbal Comprehension, with performance poorest on Comprehension, Vocabulary, and Arithmetic. The highest performing subtests were Symbol Search and Picture Arrangement.

The impairments found in individuals with FASD cannot be fully explained by a general lowering of IQ, as the deficits are present even when IQ is controlled for. Carr, Agnihotri, and Keightley (2010) studied deficits in sensory processing and adaptive behaviour in children who had been prenatally exposed to alcohol, comparing a diagnosis of FAS or pFAS with participants who have been diagnosed ARND, as well as participants who have been PAE but do not meet criteria for an FASD diagnosis. They found that impaired scores on the *SSP* and the *Adaptive Behaviour Assessment System, Second Edition (ABAS-II)* were not linked to impaired *WISC-III*

scores, with ARND participants scoring comparably to PAE individuals on IQ but significantly lower on measures of sensory processing and adaptive behaviour.

Coles, Lynch, Kable, Johnson, and Goldstein (2010) found similar results in their study of memory in prenatally exposed adolescents, for which they compared an exposed dysmorphic group and an exposed non-dysmorphic group with a matched control group and a special education contrast group. They found that despite having similar ability levels to the control group (as measured by the *Wechsler Abbreviated Scale of Intelligence* [WASI]), the exposed non-dysmorphic group evidenced significantly more memory problems. Moreover, to directly assess the hypothesis that the cognitive impairments seen in PEA individuals are secondary to global delays, Coles and colleagues analyzed their data on memory deficits controlling for Full Scale IQ (FSIQ); they found that while FSIQ was a contributor to overall performance, it did not account for the impairments they discovered.

Unfortunately, without an IQ below the cut-off for mental retardation, these individuals will have difficulty obtaining the appropriate services from their schools and communities (Streissguth et al., 2004) because many programs and services are restricted to individuals who meet the DSM-IV criteria for mental retardation. For example, in Saskatchewan individuals have to meet the DSM-IV criteria for mental retardation to be eligible for services from the Community Living Division of the Ministry of Social Services. Access to special education resources within schools is based on an internal needs-based assessment rather than by an IQ or achievement cut-off scores. It is not known how many children in Saskatchewan with an FASD diagnosis qualify for special education services.

1.1.3.4. Communication: receptive and expressive. Language consists of two components: receptive language, which is the capacity to understand language, and expressive language, which is one's ability to convey ideas in verbal form (Church & Kaltenbach, 1997). Compared to other areas of CNS dysfunction, language impairments in people with an FASD have been relatively understudied. In a study of 25 children with heavy PAE and 26 controls, all between the ages of three and five, communication skills were examined using the Clinical Evaluation of Language Fundamentals (McGee et al., 2009). The PEA participants had significantly impaired language abilities when compared to the control group on both expressive and receptive language. The PAE group demonstrated better receptive than expressive skills, but this finding was the same for non-exposed participants.

Nash and colleagues (2013) examined differences between 109 children diagnosed with FASD and 61 undiagnosed children who were prenatally exposed to alcohol. They noted differences on the language composite of the *NEPSY-II* (a developmental neuropsychological assessment), with participants with FASD scoring significantly lower than their PAE but undiagnosed peers.

Some researchers propose that the difficulty people with FASD have in acquiring language may be related to deficits in auditory processing (Stephen, Kodituwakku, Kodituwakku, Romero, Peters, et al., 2012). Complicating this area of research is a fact that many children diagnosed with an FASD in Canada are either aboriginal or are foreign adoptees. Both of these groups frequently have English as a second language.

1.1.3.5. Academic achievement. Generally, it is recognized that children with FASD will have problems of achievement. The minimal research in this area indicates that children diagnosed with FASD demonstrate impairments on math with average scores on measures of reading and spelling. Howell and colleagues (2006) found individuals prenatally exposed did not display deficits on reading or spelling as measured by the *Wechsler Individual Achievement Test (WIAT)*, but showed significant impairment on mathematics and math reasoning. Similarly, Nash and colleagues (2013) found that participants in their sample with FASD scored lower than their undiagnosed (but PAE) peers on numerical operations and math reasoning of the *WIAT*. In a study of the neuropsychological deficits present in individuals with FASD, Olson, Feldman, Streissguth, Sampson, and Bookstein (1998) compared FAS participants to adolescents with minimal or no PAE on the *Wide Range Achievement Test, Revised (WRAT-R)*. In their sample, achievement scores were consistent with expectations given the participant's ability scores, with the exception of arithmetic scores, which were significantly lower than expected. Streissguth and colleagues (2004) examined the negative outcomes of individuals with an FASD face, using a sample of 415 patients from the University of Washington's Alcohol Follow-up Study. Achievement was measured with the *WRAT-R*. They found that group means for subtests related to reading and spelling are consistent with what one would expect given their IQ scores; however, scores on tests of arithmetic are 2/3rds of a standard deviation below the average IQ. This finding is present across the diagnostic spectrum.

There are a number of possible explanations or contributing factors to impaired math performance in individuals with FASD. Several of the brain structures identified as abnormal in

people with FASD, such as the parietal and frontal lobes, have been connected to mathematical thinking (Dehaene, Molko, Cohen, & Wilson, 2004). Rasmussen and Bisanz (2010) attempted to elucidate this relationship by examining the role of working memory in math achievement. Using a sample of 41 children (21 of whom were diagnosed with FASD), they looked at scores on the *Working Memory Test Battery for Children (WMTB-C)* and two math subtests of the *Woodcock Johnson Test of Achievement, Third Edition (WJ-III)*. It was found that participants with FASD scored lower overall on the mathematical achievement measures, demonstrating particular vulnerability to quantitative concepts (such as knowledge of math concepts and symbols) when compared to the ability to solve applied problems. The FASD affected children also scored lower on the working memory measure. Not only were the measures of math ability correlated more significantly with working memory scores for children diagnosed with FASD than children without FASD, group differences in math were reduced once scores on the *WMTB-C* were used as a covariate. This suggests math performance in individuals with FASD may improve if the working memory demands of the task are reduced and lends evidence to the notion that impaired achievement may partially be a reflection of other neurocognitive difficulties.

It is difficult to determine at the moment the precise FASD deficits related to poor achievement, as there are many confounding variables that contribute to achievement success or failure. FASD is overrepresented in lower socioeconomic communities (Abel, 1998; Chudley et al., 2007) and average standard scores are poor for children of low socioeconomic status, even among those without prenatal exposure to alcohol (Howell et al., 2006). A meta-analysis of the research shows that lower socioeconomic status is related to reduced academic achievement due to such factors as the availability (or lack) of resources at home, the general school environment (including materials, instruction, and teacher-student ratio), and parental relationships with school personnel (Sirin, 2005). In one sample (Streissguth et al., 2004), 60% of the participants with FASD had undergone a disrupted school experience (DSE), defined as expulsion, suspension, or quitting. It is important to understand whether children and youths are not reaching their potential because of CNS dysfunction directly, or difficulties in the school environment related to the CNS dysfunction.

1.1.3.6. Memory. Deficits in memory for individuals with FASD appear to be complex. Such people demonstrate significant deficits in verbal learning and general memory processes (Manji, Pei, Loomes, & Rasmussen, 2009; Mattson & Roebuck, 2002). Comparing a sample of

35 alcohol exposed children to 35 control children, all between the ages of eight and sixteen, Mattson and Roebuck (2002) measured impairment in memory with the *California Verbal Learning Test (CVLT)*, the *Biber Figure Learning Test (BFLT)*, and three subtests of the *Wide Range Assessment of Memory and Learning (WRAML)*. In their sample, participants with PAE learned less information and reached a learning plateau more quickly than did controls. While the PAE group's scores for both the *CVLT* and the *BFLT* were lower than controls, the PAE group learned a similar number of new designs per trial on the *BFLT*; in contrast, PAE participants learned fewer words per trial than their control counterparts on the *CVLT*.

In addition to achievement scores, Olson and colleagues (1998) also assessed memory differences between FAS and control adolescents using a variety of measures, including Digit Span, the *Seashore Rhythm Test*, and the *Sequence Learning Task*. Their sample of adolescents evidenced greater difficulty with short-term memory when compared with the control group. While procedural memory did not appear to be impaired, declarative learning (that which requires the conscious recollection of learned information) was significantly impaired for the FAS participants. Memory for faces and numbers appears to be particularly impaired in people with FASD (Rasmussen et al., 2006). In a review of the literature on memory deficits in prenatally exposed individuals, Manji and colleagues (2009) conclude that visuo-spatial memory deficits are dominant, and span all age ranges. Children with FASD have been found to score lower than their PAE but undiagnosed peers on the *Children's Memory Scale (CMS)* (Nash et al., 2013). Specifically, participants with FASD performed more poorly on General Memory as well as on Dot Locations, both the learning and the long delay scores.

Another study used the *Rey-Osterrieth Complex Figure Test (ROCF)* to assess for visual-spatial perception, memory, and learning (Pei, Job, Kully-Martens, & Rasmussen, 2013). The *ROCF* task requires one to copy a complex design and then reproduce the same design after a short and long time delay. The sample consisted of 70 children, aged 6 to 12; 35 participants had a diagnosis of FASD and 35 were born healthy controls to mothers who abstained from prenatal alcohol use and did not experience complications during pregnancy or birth. Children in the control group demonstrated greater encoding of information, as well as less decay of design details over a delayed period. Children with FASD evidenced greater difficulty in terms of organization during initial encoding, accuracy in remembering structural details of the design as time progressed, and more consistent errors in the misplacement of design features. The authors

conclude that in this sample, the ability to recall information to mind was greatly influenced by problems in initial encoding and less by memory decay. They also noted that the participants with FASD were more likely to struggle with structural accuracy, suggesting they have more difficulty seeing the whole object when compared with healthy controls.

An area of memory particularly important for understanding the relationship between FASD and the criminal justice system is source monitoring. Source monitoring is a form of memory that requires judgments about the origin of information (Johnson, Hashtroudi, & Lindsay, 1993). It is particularly important in the subjective experience of autobiographical memory (i.e., the sensation of recalling a specific experience in one's life) and disruption in source monitoring can lead to confabulation, a problem commonly found in individuals with FASD (Fast & Conry, 2004; Fraser, 2008). In the criminal justice system, this can lead to serious problems when giving a statement or testimony, as people who confabulate may have difficulty differentiating between what they have personally experienced and what they may have read, heard, or seen on television.

In a sample of 57 children, 19 diagnosed with FASD and 38 controls, participants were asked to complete a source monitoring task that involved three conditions: reality, internal, and external (Kully-Martens, Pei, Job, & Rasmussen, 2012). Reality monitoring is the ability to differentiate between internally and externally generated information; external monitoring is the ability to distinguish between information provided by at least two separate people; and internal monitoring was defined as the ability to discriminate between two types of self-generated memories. It was found that participants with FASD demonstrated impairment in recognition and source memory compared to the control group. Children with FASD were poorer in distinguishing between old and new information, with more false alarm errors (i.e., they identified new information as old more frequently than the control group). Those with FASD remembered less information and the quality of their recalled information was poorer. The authors conclude impairment in both the organization and contextualization of incoming information impedes accurate source monitoring in people with FASD, as these difficulties cause errors in information retrieval. Children and adolescents with FASD may be viewed as liars or manipulative when their brains have constructed inaccurate memories based on cognitive impairment.

Other research also indicates that memory impairments found in FASD may stem from deficient encoding and retrieval processes (Coles et al., 2010; Manji et al., 2009). Coles and colleagues (2010) found no evidence that memory impairments in people with an FASD are due to forgetting information that had previously been encoded. Rather, they found memory deficits to be a result of impaired learning processes by analyzing the difference in scores on a verbal learning task between participants with PAE and control participants.

An alternative or complementary theory is raised by the research of Rasmussen, Pei, Manji, Loomes, and Andrew (2009). In their sample of 70 children, participants were divided by FASD diagnosis and age to assess how language and visual memory strategies are used at different developmental stages. It was found that younger children with FASD used a visual strategy whereas their peers without FASD had already moved onto a verbal memory strategy, indicating that children with FASD may develop memory strategies more slowly than those unaffected by PAE, if they develop these strategies at all. Higher executive functioning skills were related to verbal memory strategy application in the FASD condition but not in the control group (the authors note the lack of variability in the control group may be responsible for not finding a relationship between executive functioning and strategy in the healthy sample, as many younger children in the control group had already moved past visual strategies). In addition, children in the FASD condition were more likely to use “other” strategies (i.e., neither verbal nor visual) or no strategy at all. From these results, Rasmussen and colleagues conclude that the memory impairments observed in people with FASD may be reflective of a strategy problem instead of a pure memory deficit and may interact with executive functioning deficits, rendering those affected as appearing more memory impaired than is the reality. As research in this area progresses, it is probable that it will be discovered that many of these variables interact to produce the impairments present in FASD.

1.1.3.7. Executive functioning and abstract reasoning. Executive functioning is a term used to describe cognitive processes related to the frontal areas of the brain and frontal subsystems involved in planning, response inhibition, self-regulation, organization, and the integration of cognitive output processes (Archibald & Kearns, 1999). Deficits in executive reasoning are among the most consistently reported FASD impairments (Brown, Wartnik, Connor, & Adler, 2010). Generally, participants prenatally exposed to alcohol demonstrate nearly global deficits in executive functioning when compared with control participants (Green,

Mihic, Nikkel, Stade, Rasmussen, et al., 2009; Schonfeld, Paley, Frankel, & O'Connor, 2006; Rasmussen et al., 2006; Vaurio, Riley, & Mattson, 2008). Green and colleagues (2009) administered the *Cambridge Neuropsychological Tests Automated Battery (CANTAB)* to 89 children aged eight to fifteen with an FASD diagnosis, and 92 children with no PAE. The groups were matched on age. Compared to the control group, participants with an FASD diagnosis demonstrated deficits in attention (as determined by longer reaction and decision times), and impairments in planning and spatial working memory that increased with task complexity (as determined by more errors and poorer use of strategy). Differences in performance between diagnostic subgroups were minor or non-existent.

Schonfeld and colleagues (2006) examined the relationship between executive functioning deficits and daily social skills in 98 participants aged six to eleven. Each of the participants had been exposed prenatally to alcohol. Using scores on the *Behaviour Rating Inventory of Executive Functioning (BRIEF)*, as rated by their parents, Schonfeld and colleagues determined that executive functioning was impaired across all diagnostic subcategories; group differences were not statistically significant, similarly to the findings of Green and colleagues (2009).

In comparing participants with prenatal alcohol exposure to non-PAE participants with Attention Deficit/Hyperactivity Disorder, as well as non-exposed controls, Vaurio and colleagues (2007) looked at scores on the *Wisconsin Card Sorting Test (WCST)*, the *Controlled One Word Association Test (COWAT)* and the *Trails Making Test (TMT)*. In their sample, participants with PAE demonstrated impairments on all tasks when compared with healthy controls, with the exception of the simplest *Trail Making Test (TMT-A)*. The PAE participants' pattern of deficits differed from those found in the ADHD group; those exposed to alcohol scored significantly lower on tests of letter fluency, and displayed a relative impairment on the more complicated trail making task (*TMT-B*) when compared to *TMT-A*. Those with ADHD evidenced greater impairment on the *WCST*.

Deficits in response inhibition are found repeatedly in samples of people with FASD when compared to individuals with no prenatal exposure to alcohol. Mattson, Goodman, Caine, Delis, and Riley (1999) looked at executive functioning in four areas: cognitive flexibility, response inhibition, planning, and concept formation and reasoning. Their sample consisted of 28 participants: eighteen with a history of heavy prenatal alcohol exposure (with or without an

FASD diagnosis), and ten non-exposed controls. They measured the aforementioned areas of functioning using subtests of the *Delis-Kaplan Executive Function Scale (D-KEFS)*, specifically the Trail-Making Test, the Stroop Test, the Tower Test, and the Word Context Test. Mattson and colleagues noted that PAE participants demonstrated impairment in response inhibition when compared with controls, as determined by their responses on the Tower Test (passed fewer items and broke more rules) and the Stroop Test (more errors and longer recorded times). Similar to previous findings, differences between subgroups of alcohol-exposed participants were not statistically significant.

From the research, it can be concluded that individuals with FASD evidence difficulties in planning, strategy use, response inhibition, organization, and cognitive flexibility (Green et al., 2009; Mattson et al., 1999; Olsen et al., 1998; Schonfeld et al., 2006; Vaurio et al., 2007). It is also important to note that executive functioning impairments do not vary by FASD diagnosis, indicating that those without the physical manifestations of the disorder evidence similar levels of executive dysfunction to those with a diagnosis of FAS (Chasnoff et al., 2010; Green et al., 2009; Mattson et al., 1999). A recent study conducted by Nash and colleagues (2013) found no significant differences between participants with and without an FASD on any measure of executive functioning in a sample who had all been exposed to alcohol in utero. It may be that executive functioning deficits are present in individuals with PAE, regardless of a formal FASD diagnosis, or it may be that deficits in executive functioning do not distinguish people with FASD from other clinical populations. The authors also suggest that executive functioning impairments are not easily captured in a laboratory setting, although this would be an issue for all research employing standardized measures of executive functioning, not strictly research into FASD deficits.

Abstract reasoning in individuals with FASD has received little empirical attention. Research suggests that people prenatally exposed to alcohol demonstrate some impairment in abstract thinking. In their sample of 28 children (18 alcohol exposed and 10 controls), Mattson and colleagues (1999) used the Word Context Test of the *D-KEFS* to assess concept formation and reasoning. They determined that participants with PAE had deficits in analytical thinking, as their responses on this measure were impaired. Participants with PAE also made significantly more target-to-nontarget responses, which are errors in which an incorrect response follows a correct response. The PAE group differed from the control group, but differences between PAE

diagnoses were insignificant. As research in this area is limited, scholars call for further investigation before conclusions regarding abstract thought are drawn (Mattson et al. 1999).

To date, research has focused almost exclusively on what are known as “cool” executive functioning (Kerr & Zelazo, 2004). Cool executive functions are those that are cognition-based and thought to be typically invoked when the abstract manipulation of information is required. Kully-Martens, Treit, Pei, and Rasmussen (2013) examined the role of “hot” executive functions in people with FASD; hot executive functions are those believed to include emotion and motivation, with actions based on reward and punishment (Kerr & Zelazo, 2004). The authors argue that rarely are decisions made without an emotional or motivational component so to observe strictly cool functioning ignores an important part of the picture (Kully-Martens et al., 2013). They administered the *Iowa Gambling Task (IGT)* on a sample of 62 children, 31 of which had been diagnosed with FASD. The *IGT* is a measure of hot executive function on which healthy people show a learning curve. It was found that participants with FASD performed significantly worse overall on the *IGT*, with a flat and generally random learning curve (Kully-Martens et al. 2013). The non-PAE peers demonstrated a positive learning curve. This finding indicates that children and youth with FASD may not be as sensitive to reward or punishment and future consequences than healthy controls, are likely more vulnerable to negative influences, and may make the same errors repeatedly. Thus poor decision-making may be more indicative of impaired hot executive functions than deviance, conduct issues, or an intentional lack of concern. This may be tied to impaired regions of the brain, as damage to the prefrontal cortex has been found to result in inappropriate social behaviour, lack of concern for others, and poor social decision-making in individuals with otherwise normal cognitive abilities (Kully-Martens et al., 2012). In addition, participants with FASD did not improve in their decision-making with age group, whereas healthy controls become more adept (Kully-Martens et al., 2013). From their findings, the authors deduce that the deficits present in FASD may become more pronounced as the youth ages, when compared to typically performing peers, due to an increase of expectations and more complex social situations. However, this question could not be answered by their data set as it was not longitudinal.

1.1.3.8. Attention. Deficits in attention are nearly always present in individuals with FASD, but the impairments are not global in nature, nor is the exact nature of these deficits yet clear.

On the attentional subtests of the *CANTAB*, participants with FASD registered longer reaction and decision times than age and sex-matched controls, with insignificant differences amongst the subdiagnostic categories (Green et al., 2009). Mattson, Calarco, and Lang (2006) examined focused and shifting attention in participants with a history of heavy PAE, using a sample of 40 children aged nine to fourteen years divided into participants with PAE and a healthy control group. They measured attention with a computerized task developed to evaluate three conditions: visual focus, audio focus, and auditory-visual shift. The participants who were prenatally exposed to alcohol evidenced significant deficits in visual focused attention, with poorer accuracy and diminished reaction times. In auditory conditions, accuracy scores remained low but reaction times only differed from controls at the longest interval. This suggests that sustaining attention over extended intervals (defined as greater than ten seconds) is a particular challenge for people with an FASD.

Contrary to this, Connor, Streissguth, Sampson, Bookstein, and Barr (1999) found different results in their sample of FASD-diagnosed adults taken from the University of Washington's Fetal Alcohol Follow-Up Study. Four tests were used to assess for visual and auditory attentional functioning: the *Letter Cancellation Test (LCT)* and *Continuous Performance Task (CPT)* to measure sustained attention and focusing, and the *Consonant Trigrams Task (CTT)* and *Attentional Process Test (APT)* to measure auditory attention. Compared to age-matched controls, they determined that while participants with FASD demonstrated impairments in both auditory and visual attention tasks, the strongest deficits were found in auditory attention. Connor and colleagues noted that the most discriminating tests for deficits in FASD were all auditory tasks (the *CPT* tone task, *APT*, and *CTT*).

Clearly, the specific attentional deficits associated with FASD have yet to be fully clarified by the research. However, it does appear that deficits in attention are present (Connor et al., 1999; Green et al., 2009; Mattson et al., 2006), and that such individuals are likely to struggle with sustained attention (Mattson et al., 2006).

1.1.3.9. Adaptive behaviour, social skills, and social communication. Children and youth with FASD are often seen as having impaired social functioning. Using a sample of 33 children with FASD, 30 children with ADHD, and 34 normal controls, Greenbaum, Stevens, Nash, Koren, and Rovet, (2009) explored social cognition and emotional processing abilities. All children were aged six to thirteen, and were rated by their parents on the *CBCL* and their teacher

on the *Teacher Report Form (TRF)*. Both parents and teachers rated each participant on the *Social Skills Rating System (SSRS)*. Social cognition was measured using three tests from a previous social cognition battery (Saltzman, Benaiah & Lalonde, 2007): False Belief, Intention, Deception and Sarcasm; Interpretive Theory of Mind; and Strategic Control of Emotions: Using Display Rules. They used four subtests from the *Minnesota Test of Affective Processing (MNTAP)* to assess for emotional processing by testing for emotional recognition and discrimination in faces and samples of speech. Parents and teachers rated children with FASD as having more total behavioural problems than children with ADHD or controls on the *SSRS*, although only teachers rated the FASD group as having greater externalizing issues than ADHD; parents rated ADHD and FASD participants as equally affected (Greenbaum et al., 2009). The parents of children with FASD rated their social skills in the average range, whereas teachers rated participants' with FASD social skills as deficient. This discrepancy is not unusual, and can be attributed to such confounding factors as size of comparison groups (teachers having a much greater pool of reference), and interaction situations (teachers see the child struggle with learning, or functioning in a structured setting; Greenbaum et al., 2009). On the measures of social cognition and emotional processing, significant deficits were found in children with FASD (Greenbaum et al., 2009). Participants with FASD demonstrated lower facial and auditory emotion processing skills than controls and participants with ADHD, as well as lower scores on the Display Rules measure of social cognition but not False Beliefs or Theory of Mind. Thus it appears not all aspects of social cognition are impaired in those prenatally exposed to alcohol.

In addition to assessing executive functioning in children exposed to alcohol in-utero, Schonfeld and colleagues (2006) asked the parents and teachers of their 98 PAE participants to complete the *SSRS* in order to assess social impairments in people with an FASD. They found social skills were rated by both parents and teachers as clinically impaired across the FASD spectrum, with no statistically significant differences between groups.

In a longitudinal project involving 158 FAS children first assessed between 1977 and 1991, Steinhausen and Spohr (1998) examined the long-term outcome for those prenatally affected by alcohol. The parents of participants (or their legal guardian) were asked to complete the *CBCL* and their teachers were asked to fill out the *TRF*. The results show that after attentional difficulties, social relational problems were observed to be the most frequently reported concern in children with an FASD.

An analysis of social skills functioning in children with PAE examined teacher and parental ratings on the *SSRS* for a sample of 60 children, 37 of whom had been prenatally exposed to alcohol (Rasmussen, Becker, McLennan, Urichuk, & Andrew, 2010). In comparison to the non-exposed control group, children with PAE demonstrated deficits on the caregiver's ratings of responsibility, hyperactivity, internalization problems, and overall social skills. It was found that the social skills deficits were present even once behaviour problems were held constant. The authors also determined that poor social skills were not purely accounted for by living away from one's biological family, as scores on the *SSRS* were unrelated to length of time in current home and the number of previous placements.

McGee, Bjorkquist, Price, Mattson, and Riley (2009) looked at social information processing in children aged seven to eleven. 26 children with a history of heavy PAE were compared with 26 normal control children, matched on age, sex, socioeconomic status, and race. Abilities in social information processing were assessed using the *Social Information Processing Interview* and the *Test of Problem Solving, Third Edition Elementary Version (TOPS-3)*. Compared to the control group, individuals with FASD demonstrated greater difficulty with such processes as goal selection, response generation, and response evaluation. They also evidenced deficits in encoding and attribution. Regarding the *TOPS-3*, the FASD group performed significantly poorer than controls across all subscales: Making Inferences, Predicting, Determining Causes, Sequencing, Negative Questions, and Problem Solving.

Finally, Nash and colleagues (2013) examined differences in their sample of children with PAE, to see if children diagnosed with FASD evidenced impairment compared to PAE children without a diagnosis of FASD. On the caregiver form of the *CBCL*, participants with FASD were noted to have higher reported internalizing, externalizing, and total problems. Caregivers were also asked to complete the *BRIEF* and *Connor's Rating Scale (CRS)*; children with FASD were rated more highly on the Behavioural Regulation Index, Metacognition Index, and Global Executive Composite on the *BRIEF* and as having higher ADHD, Global, and DSM Total scores. The teachers were asked to complete the same measures, only using the teacher's form, and the results were almost identical as for the caregivers. The one distinction between teacher and caregivers ratings was that teachers did not report any differences between children diagnosed with FASD and children not diagnosed with FASD on the *BRIEF*. This may be due to the greater structure and rules in a classroom when compared to home life or the use of

medication while children are at school. Overall, the authors note that children with FASD had greater internalizing, externalizing, and attentional problems than their PAE peers. This suggests a combination of an increased risk for externalizing behaviour paired with low inner monitoring and poor planning abilities.

The deficits in social skills found in people with FASD have been linked to the impairments in executive functioning. Schonfeld and colleagues (2006) determined that the neurocognitive impairment in executive functioning were predictive of their participants' social behaviour, as measured by the *SSRS*; both teachers' and parents' ratings of social skills was predicted by parental rating scores on the *BRIEF*. In addition, impaired social functioning also appears to be related to deficits in sensory processing. In their aforementioned study, Franklin and colleagues (2008) found a significant relationship between sensory processing deficits and problem behaviours, and suggested that the deficits found in sensory programming in children with FASD may impact their ability to adaptively respond to their environment. Kully-Martens, Denys, Treit, Tamana, and Rasmussen (2012) hypothesized that sensory processing may also contribute to social difficulties by increasing emotional lability and hyperactivity. Another possible explanation is that social skills impairment is tied to cerebral damage in a number of areas, including the amygdala, orbitofrontal cortex, corpus callosum, and temporal cortex (Kully-Martens et al., 2012).

Individuals with FASD also demonstrate significant deficits in adaptive behaviour. In a sample of 415 taken from the University of Washington's Fetal Alcohol Follow-Up Study, adaptive behaviour was measured by ratings on the *VABS*; responders included biological and adoptive parents, as well as step- and foster parents, other relatives, and caregivers (Streissguth et al., 2004). The results show that the mean scores for people with an FASD are significantly lower than the general population, and between 1 and 1.5 standard deviations below their mean IQ scores.

Jirikowic, Kartin, and Olson (2008) used a sample of 25 children with an FASD and 23 normal controls (aged five to eight) to obtain a descriptive profile of adaptive function in FASDs. The participants' primary caregiver completed the *Scales of Independent Behaviour Revised (SIB-R)* to provide information on the child's adaptive and problem behaviours across home, school, work and community settings. On caregiver reports, children diagnosed with FASD were rated lower than control children in areas of personal living skills, social interaction and

communication, and community living skills. As well, they were rated higher in maladaptive behaviours, with more than three quarters of the FASD group classified as having marginally serious to serious behavioural concerns.

Impairments in adaptive behaviour do not appear to be correlated with IQ scores across the diagnostic spectrum. Carr and colleagues (2010) used a sample consisting of 48 children: 15 with PAE, 16 with a diagnosis of ARND, and 15 with a diagnosis of either FAS or pFAS (dysmorphic). Participants were rated on the *SSP* and the *ABAS-II* by a parent; the majority of respondents were a foster parent to the participant. They found that participants with ARND had intelligence scores comparable to those in the PAE group, but performed significantly worse regarding adaptive functioning. In fact, those with ARND scored significantly lower on the *ABAS-II* than did participants with PAE or pFAS/FAS, lending credence to the theory that the number of the deficits present in ARND are no less serious than is found in FAS or pFAS, and may even be more pronounced due to the lack of early recognition and intervention.

Akin to social functioning, adaptive behaviour skills seem to be related to deficits in sensory processing. Carr and colleagues (2010) examined the relationship between sensory processing and adaptive behaviour using correlational analysis. They found a significant positive correlation between ratings on the *SSP* and the overall adaptive behaviour score of the *ABAS-II*, as well as the practical domain, suggesting a relationship between a child's capacity to process sensory information and the adaptive behaviours they use as a reaction.

1.1.4. CNS Dysfunction Defined

Chudley and colleagues (2005) made significant advances by standardizing the diagnosis of FASD across Canada, yet they did not provide information as to how to operationally define the constructs of interest. For example, while cognition as a term refers to one's general cognitive abilities, we cannot directly measure their cognitive skills or intelligence. Particular psychological measures representative of each domain are instead used to assess said domain; this is operationally defining the construct by its test results.

Recent research has defined executive functioning in individuals with FASD by scores on the *CANTAB* and the *D-KEFS* (Mattson, Roesch, Fagerlaund, Autti-Ramo, Jones, et al., 2010a; Mattson, Foroud, Sowell, Jones, Coles, et al., 2010b). Cognitive ability has been defined as the results on the *Leiter International Performance Scale, Revised* (*Leiter-R*; Mattson, Roesch, Fagerlaund, Autti-Ramo, Jones, et al., 2010a; Mattson, Foroud, Sowell, Jones, Coles, et al.,

2010b), the *WJ-III* (Lebel et al., 2010), the *WISC-IV* (Mattson et al., 2010b), and the *WASI* (Coles, Lynch, Kable, Johnson, & Goldstein, 2010). Attention has been defined by the *Neurobehavioral Evaluation System, Third Edition (NES3) Continuous Performance Task* (Mattson et al., 2010b). Memory has been represented by the *Verbal Selective Reminding Memory Test (VSRT)* and the *Nonverbal Selective Reminding Memory Test (NVSRT)* (Coles et al., 2010), and memory subtests of the *CANTAB* (Mattson et al., 2010b). Deficits in adaptive behaviour have been defined as deficient scores on the *VABS* (Crocker, Vaurio, Riley, & Mattson, 2009).

1.1.5. The Low Exposure Consideration

As mentioned previously, medical, psychiatric and psychological researchers advocate for complete abstinence during pregnancy, stating that there is no known safe amount to drink (Arendt & Farkas, 2007; Astley, 2006; Sokol et al., 2003). The official stance of the Interagency Coordinating Committee on FASD (ICCFASD) is that the teratogenic effects of alcohol on the brain can occur with a variety of alcohol doses throughout pregnancy (2011). This is because it appears that a number of variables contribute to the development of FASD beyond the mere ingestion of alcohol, including the mother's nutrition and body's ability to metabolize alcohol (Abel, 1998). Another factor hypothesized to influence whether a fetus develops FASD is the extent of prenatal alcohol exposure. Some researchers argue that there is no evidence that low prenatal exposure results in impairment of the fetus (Kelly, Sacker, Gray, Kelly, Wolke, et al., 2009a; Kelly, Sacker, Gray, Kelly, Wolke, et al., 2010; O'Leary & Bower, 2012).

However, much of the research in this area has been done on children who are too young to demonstrate difficulties. There is evidence in the literature to suggest that differences between PAE children and non-PAE children become more pronounced with age, with many of the deficits not apparent prior to attending school (Kelly-Martens et al., 2013, Zelnor et al., 2012). Zelnor and colleagues (2012) found this to be the case in their study of a single case with heavy PAE: at three months, a neurodevelopmental assessment revealed no delays; slight delays in motor development were detectable at ages six and eight months; by 14 months, delays in motor and language skills were apparent, particularly gross motor abilities and expressive language skills. Keeping in mind that this was a single case study, even children who are exposed to heavy PAE may be likely to appear more like their healthy peers when they are young. The rates of secondary disabilities increase as people with FASD age, often because treatment plans fail to

take into account the heightened risk of substance abuse, school failure, and need for assistance as adults (Burd, Fast, Conry, & Williams, 2010). To date, the majority of research on low exposure to alcohol in utero has consisted of preschool samples (Kelly et al., 2009a; Kelly et al., 2010).

In addition, not all research conducted on low PAE pregnancy finds low exposure to be without risk. Using data taken from the United States National Longitudinal Survey of Youth, based on a sample of 1618 participants, it was determined that low to moderate and high prenatal exposure to alcohol both resulted in infant difficulties (Chen, 2012).

This controversy does not suggest the absence of hope in determining if there is a safe amount for a woman to drink while pregnant. A large confounding factor in low exposure research is the lack of consistency in how “low exposure” is defined (Moyer, 2013); what is a low dose in one sample may be considered moderate drinking in another study. Additionally, if the researchers have not specified drinking patterns, one drink per day for a week would be classified similarly to a binge episode of seven drinks. This is an important factor as it becomes increasingly probable that the deficits associated with FASD are related to a binge-drinking pattern (Fraser et al., 2012; Hepper et al, 2012; Meyer-Leu et al., 2011). In a sample of 84 pregnant women in Ireland, Hepper and colleagues (2012) divided fetuses into groups based on dose of exposure and drinking pattern of the mother. Their focus was on assessing the number of trials it took the fetus to habituate to an intrauterine stimulus. They discovered the greatest impairment in children who were exposed to heavy binge drinking; moderate drinking spread evenly throughout the week did not have an affect on trials to habituate. Likewise, Meyer-Leu and colleagues (2011) found binge drinking was associated at a trend level with neonatal asphyxia in their sample of 1258 expectant mothers. Fraser and colleagues (2012) examined outcome areas such as prenatal growth, visual acuity, and cognitive development in an Inuit sample for whom it was common to binge drink on occasion, but for whom alcohol is rarely consumed otherwise. They determined that even infrequent binge drinking was associated with diminished prenatal growth and reduced visual acuity at six months old. It is valuable to note that, like much of the research on low-dose exposure, these studies were performed far too early in development to be able to assess for the cognitive and behaviour problems typically associated with FASD; further research will be required to determine the impact of binge drinking on functioning in late childhood and adolescence.

Unfortunately, doctors are still unable to account for how such factors as maternal nutrition, health, and genetic factors (such as one's body's ability to metabolize alcohol) impact the development of the disorder (Abel, 1998), and some research evaluating the pattern of PAE exposure in FASD has found no evidence of a dose threshold (Feldman, Jones, Lindsay, Slymen, Klonoff-Cohen, et al., 2012). After conducting a meta-analysis of low, moderate and heavy exposure literature, O'Leary and Bower (2012) conclude that even though there is no firm evidence for impairment from low doses of PAE, the margin of error for low dose exposure is so small that it would be unethical for the medical profession to approve of maternal alcohol consumption. Thus, although there has been some encouraging research, total abstinence remains the official recommendation to pregnant women. However, it would be beneficial if the message of abstinence were presented in an unprejudiced way so as to minimize unintended consequences, such as pregnant women with substance abuse issues avoiding treatment because of fear of stigma (O'Leary & Bower, 2012).

1.2. FASD and the Legal System

Of the secondary disabilities characteristic of FASD, the increased likelihood of involvement in the legal system is of particular current concern. Researchers suggest that individuals with FASD are at an increased risk for criminal activity and victimization due to the CNS impairments related to the disorder. Unfortunately, there has been almost no empirical research on FASD and offending behaviour completed and published at this time to inform opinion, leaving researchers to infer from what literature is available.

Scholars have hypothesized that criminal behaviour is a result of the vulnerability of an individual with FASD to manipulation and coercion, resulting from their impaired social cognition and poor social competence (Dagher-Margasian, 1997; Keil, Paley, Frankel, & O'Connor, 2010). Others have posited that criminal behaviour is more linked to executive functioning deficits, such as poor judgment and impulsivity (Fast, Conry, & Loock, 1999). Once involved in the justice system, it is expected that, due to the impaired reasoning and poor social skills found in people with an FASD, they will have difficulty judging "friends" from "strangers" (Abel, 1998; Burd et al., 2010). Anecdotally, people who work with individuals with FASD report that they are people-pleasers and are trusting of people in authority (Abel, 1998); as such, they are suspected to be more likely to confess to crimes they did not commit (Dagher-Margasian, 1997; LaDue & Dunne, 1997). In a review of the literature on communication

difficulties in people with an FASD, it was found that such individuals consistently demonstrate a discrepancy between their expressive verbal skills and their actual level of comprehension, which often gives the impression that they are more capable than they are (Akbarian, 1992; Brown et al., 2010). Dagher-Margasian (1997) argued that this could lead to inflated expectations of their abilities, particularly their ability to comprehend the legal system. The memory encoding problems (Coles et al., 2010; Manji et al., 2009) and attentional deficits (Green et al., 2009; Mattson et al., 2006) people with FASD show are thought to impact their capacity to learn from past behaviour, and impaired abstraction skills (Mattson et al., 1999) impede their ability to generalize from past situations to make cause-effect connections (Boland, Burrill, Duwyn, & Karp, 1998). There is no evidence that they are capable of learning by standard methods of punishment, such as incarceration (Barnette, 1997; Dagher-Margasian, 1997; Canadian Bar Association, 2010). In fact, it appears that people with FASD are generally insensitive to punishment and reward when compared to their non-alcohol exposed peers (Kully-Martens et al., 2013).

A recent case in Edmonton, Alberta, lends some empirical evidence to the theorizing. In a case in which two teenagers were charged with homicide, statements made by the accused were ruled inadmissible by a judge due to mistakes made by the RCMP officer in establishing the youths' rights (Keil, 2011). One of the youths had been previously diagnosed with FASD and ADHD; his lawyer left the station before the start of the interrogation and his guardian stated that the youth did not understand his lawyer's instructions in making a statement without the lawyer present. In addition, the youth had not waived his right to have a lawyer present at the time of the interview. Remembering instructions from one's lawyer is one way the memory problems present in FASD may contribute to difficulties in the legal system (Burd et al., 2010). This is a case in which valuable testimony was lost on a homicide case as a result of a youth not properly understanding his rights, due to both police negligence and likely neurocognitive impairment, and highlights the importance of examining the relationship between the deficits observed in people with FASD and the criminal justice system.

One study directly linking FASD with criminal behaviour was conducted with the assistance of the University of Washington, using 415 participants enrolled in the Drug and Alcohols Unit's Fetal Alcohol Follow-Up Study (Streissguth, Bookstein, Barr, Sampson, O'Malley, et al., 2004). Data on the social consequences of FASD was obtained through

telephone interviews with informants (i.e., parents or guardians) who completed the Life History Interview for each participant. An individual was considered to have had trouble with the law if s/he was ever charged, arrested, or convicted of an offense. In their sample, they determined that 14% of children and 60% of adults/adolescents had come into contact with the justice system. Respondents reported that 45% of crimes committed by adults and adolescents with an FASD were those committed against persons, including shoplifting/theft, assault, burglary, and domestic violence.

In addition, some empirical research has been done regarding delinquency in prenatally exposed adolescents, which is particularly relevant to the present research project. Schonfeld, Mattson, and Riley (2005) looked at moral maturity in individuals aged ten to eighteen with a history of PAE, and its relationship to delinquency. They compared a sample of 27 participants with PAE to 29 age, gender, handedness, socioeconomic status, and ethnically matched, non-exposed control individuals. Moral maturity was measured with the *Sociomoral Reflection Measure – Short Form (SRM-SF)*, and delinquency was determined through self-report on the *Conduct Disorder Questionnaire*. They found that alcohol exposed youth overall displayed lower moral maturity compared to non-exposed controls. Scores on the *SRM-SF* indicated that the PAE group's primary concern was for minimizing the negative consequences to themselves, whereas the control group was concerned for others, as well as what is socially normative. Alcohol exposed participants also admitted to engaging in significantly more delinquent behaviour than controls. However, delinquency was predicted by social desirability (as measured by the *Marlowe-Crown Social Desirability Scale*), suggesting that the participants may have under-reported behaviours, and leaving open the interpretation that PAE individuals are simply more likely to admit to delinquent behaviours than non-exposed youth.

Yet other studies that do not rely on self-reported delinquent behaviours reveal similar results of increased delinquency in alcohol-exposed youth. Disney, Iacono, McGue, Tully, and Legrand (2008) examined the rates of CD in prenatally exposed adolescents using a sample of 1252 individuals with and without PAE who were assessed at age seventeen as part of a larger study (Minnesota Twin Family Study). Diagnoses were assigned by interviewers with extensive training and at minimum a Bachelor of Arts in psychology, and the symptoms of CD were assessed through parental interview. In addition, PAE information was obtained through parental report. The youth who were prenatally exposed demonstrated significantly more symptoms of

CD than those who were not exposed to alcohol in-utero, even when parental alcoholism, parental drug abuse or dependence, and externalizing disorders were held constant. In addition, rates of CD diagnosis were 44.1% for the children of mothers who admitted to drinking, compared to 20.2% of those whose mothers did not drink while pregnant.

The systematic evaluation of FASD in offending populations is very difficult, as inmates rarely enter the judicial system with a diagnosis. Diagnosis in adults is difficult as facial features appear to change with age (Aase, 1994; Burd, Fast, Conry, & Williams, 2010; Conry & Fast, 2000; Streissguth et al., 1991), information regarding prenatal exposure to alcohol can be particularly difficult to obtain (Burd et al., 2010), and screening measures with good reliability and validity are not yet available (Boland, Chudley, & Grant, 2002). Attempts to assess for rates of FASD in offending populations suggest that individuals with FASD are overrepresented in the judicial system.

Fast, Conry, and Loock (1999) examined the occurrence of FASD in remanded youth in a British Columbia inpatient facility. Their sample consisted of all youth remanded to the psychiatric centre for an assessment for a one-year period. Each youth was medically assessed with a physical exam and completed the age-appropriate Wechsler measure of intelligence as part of a psychological evaluation; they were diagnosed as having FAS, pFAS, or ARND by a pediatrician with FASD experience. In total, 287 youth underwent the examination during the year of study, which Fast and colleagues note was 2.5% of detained youth in BC and the Yukon throughout this period of time. Fast and colleagues (1999) reported that 67 youth (or 23.3% of the sample) were assigned an alcohol-related diagnosis: one percent was diagnosed as full FAS, whereas 22.3% were diagnosed as pFAS or ARND. Only three of their participants had received a prior diagnosis of an FASD. This finding of 23% incidence for FAS is much higher than that found in the general population, which was estimated to be approximately nine babies per 1000 live births in Canada in 2003 (Alberta Health Services, 2010). As such, Fast and colleagues (1999) concluded that their findings support the hypothesis that people with FASD are overrepresented in correctional settings.

In a systematic review of the correctional literature available on prevalence estimates of FASD, Popova, Lange, Bekmuradov, Mihic, and Rehm (2011) examined six studies; five from Canada and one conducted in the United States. These represented the extent of the research on FASD in corrections, at the time of their review. Using this data, they estimated that the number

of young offenders with FASD who are in Canadian Corrections on any particular day ranged from 207 to 423; of these numbers, it was expected that only 19 (or 1%) would meet criteria for FAS. The same study estimated 3686 adults with FASD were in the prison on a given day, which is approximately 9% of adult offenders in Canadian correctional institutions. They deduce from these numbers that on any given day in a specific year, youth with FASD are 19 times more likely to be in prison than youth without FASD.

Popova, Stadel, Lange, and Rehn (2012) have extended that line of work to include total yearly cost of corrections among people with FAS specifically; this information was based on data from Statistics Canada regarding number of adult and youth offenders with FAS in the correctional system and information from the Canadian Centre for Justice Statistics on annual cost of incarceration. It is believed that the youth justice costs are between \$1.2 million and \$2.2 million per year, whereas individuals with FAS are thought to cost adult corrections \$21.8 million to \$43.4 million annually. They approximate between \$22.9 and \$45.7 million is spent yearly on corrections for youth and adults with FAS. This figure does not include the estimated rates for offenders with pFAS or ARND, and is thus likely a drastic underestimation of the true costs of the disorder on our justice system (Burd et al., 2010).

Recently, FASD has been formally recognized as a responsivity issue in regards to sentencing and intervention. In a local youth court decision, Judge M.E. Lafond (2001) criticized the justice system for continually ignoring the FAS diagnosis assigned to said youth, applying the same behavioural standards to him as to unaffected youth, and failing to raise issues of fitness to stand trial. She noted that although the youth was diagnosed with FAS at age three, no amendments had been made within the system to accommodate his needs, as though the diagnosis was of no consequence. This youth had dealt with five lawyers throughout his involvement with the criminal justice system, and none had raised concerns about his fitness to stand trial, even though a current assessment conducted for the court indicated that he could not comprehend the concepts of “guilt” and “innocence”. She stated, “One cannot but question what social policy is served by the use of the hard penal machinery of the criminal justice system to deal with the most chronic mentally disabled youth of our society” (pg. 9; 2001). Such rulings demonstrate that some professionals in the justice system are aware of the problem of FASD facing corrections, and recognize the need to alter standard practices in order to maintain fairness in judicial proceeding. In addition to the advocacy of Ms. LaFond, the Canadian Bar Association

(CBA; 2010) has declared that, as those with FASD are entitled to non-discrimination on the basis of their disability, they urge the government on all levels to amend sentencing laws for offenders with FASD and to develop policies designed to enhance the quality of life for individuals with FASD. It is their hope that such measures will stem the overrepresentation of such disabled people in the penal system.

While the importance of considering FASD in offending populations has been recognized by some (CBA, 2010, Lafond, 2001), many legal professionals' understanding of the disorder is limited, and few have made significant changes in their practice to accommodate the disorder and its consequences (Paley & Auerbach, 2010). A review of legal cases from the Legal Issues Resource Centre found that FASD was raised as a relevant defense issue in approximately 100 US cases in the previous 20 years (Brown et al., 2008). This result suggests that many individuals with FASD were convicted every year without awareness that they had brain damage, or that FASD was often not recognized by the court professionals as having significance on the criminal case. Unfortunately, when determining the relevance of neurocognitive impairment to a criminal case, often only IQ is considered. Thus, many legal professionals suffer from the misconception that individuals with FASD who have an average IQ have corresponding functional capacity, when it is known that low IQ does not account for the various neurocognitive deficits observed in FASD (Carr et al., 2010; Coles et al., 2010). More important to meeting the criteria for social responsibility are scores of adaptive functioning (Brown et al., 2010), which have been found to be 1 to 1.5 standard deviations below one's mean IQ score (Streissguth et al., 2004).

In an attempt to better understand the current legal culture's knowledge of FASD, Cox, Clairmont and Cox (2008) administered a survey questionnaire to 20 New Brunswick Provincial Judges and 19 Crown Prosecutors; the survey queried their awareness of FASD and its impact on their practice, as well as basic FASD knowledge, views on the disorders, and future training needs. While 95% of participants responded that they were aware of FASD, 53% reported the mass media as their sole source of information regarding the disorders, although 69% of judges obtained their knowledge through professional training workshops. 75% of judges and 70% of prosecutors based their suspicion of FASD on the physical features (Cox et al., 2008). This is of concern, as the majority of people with FASD show few if any of the physical features (Chudley et al., 2007; May et al., 2004), and it is more probable that those without physical features will

go without a diagnosis, increasingly their likelihood of becoming involved with the justice system (Loock, Conry, & Fast, 2005).

Only 26% of prosecutors and 17% of judges reported that they would send an individual expected of having an FASD for a professional assessment (Cox et al., 2008). However, the same legal professionals claim they have little access to diagnostic services or trained support professionals, meaning this statistic may not accurately represent how they would ideally operate their practice. In addition, many seemed completely unaware of their options, querying what treatments and services are available. Perhaps most alarming was the lack of knowledge around FASD symptoms and terminology; only three respondents (all judges) were aware that FAE is no longer a clinical term used in diagnosis, and only one was familiar with the diagnosis of ARND. On a more positive note, the vast majority of legal professionals recognized the ill affects of prenatal alcohol use on the fetus and agreed that diagnosis would lead to more suitable consequences, while denouncing the ideas that FASD is only a problem in youths and minority families. Thus it would appear that legal professionals are willing to incorporate changes into their practices and acknowledge the impact FASD has on an individual, but problems of training and general knowledge of the disorder still remain.

The issue of criminal behaviour in people with FASD is not without controversy. Malone and Koren (2012) argue that the majority of research in the area is conducted on forensic or clinical samples, which are likely to come to clinical or legal attention due to problematic behaviours. They argue such research does not consider postnatal factors that contribute to risk, such as low socioeconomic status, adoption, family stress, childhood physical and sexual abuse, a convicted parent, and parental substance abuse; often children exposed to alcohol in utero are at greater risk to experience these detrimental postnatal factors. They also note that children exposed to alcohol in utero often suffer a number of other prenatal risk factors, such as genetic predisposition for behaviour problems. The authors conclude that the behaviour problems seen in children with FASD may not be due to the teratogenic effects of alcohol but the associated genetic and environmental variable that confound the relationship.

It cannot be denied that all the abovementioned features likely contribute to offending behaviour in people with FASD. However, the complaint ignores that recent literature has made good efforts to account for these factors. Jones and Streissguth (2010) note that their research controlled for socioeconomic status, maternal education, race, maternal age, parity, marital

status, and religious affiliation. However, they acknowledge that they were unable to control for childhood abuse, parental substance use, or parental/familial discord. Larkby, Goldschmidt, Hanusa, and Day (2011) examined the data from a longitudinal sample of 1345 participants. This sample consisted of a cohort of women who drank while pregnant, an average of 3 drinks a week, and a cohort who drank less or abstained from substances. Held constant were the following factors: prenatal exposure to tobacco, marijuana, cocaine, and other illicit drugs; income; race/ethnicity; gender; parenting style; life events; home environment; family history of alcohol problems; and maternal lifetime psychopathology. Even with these covariates factored out, adolescents in the alcohol exposed group were three times more likely to meet criteria for lifetime diagnosis of conduct disorder than the youth whose mothers drank little or abstained.

1.2.1. The Youth Justice System

This research question is not only important in helping account for criminality in people with FASD, it also calls to question the interventions and punishments being used on adolescent populations. The current literature suggests that the incarceration of youth does not reduce recidivism and may increase rates of offending (Aizir & Doyle, 2013; Robinson, 2013a). In Australia, the jailing of juveniles has been used extensively and is failing to reduce crime rates in youth populations (Robinson, 2013a). Up to 70% of the youths being jailed in Australia are Aboriginal, and Aboriginal youth are 40 times as likely as non-Aboriginal youth to be incarcerated. Malcolm McCusker, the governor of West Australia, has stated that the mandatory jailing of juveniles may lead to “unintended consequences” that end up punishing vulnerable children in need of intervention and community support. Western Australia’s Legal Service also warned policy and law makers that children who are vulnerable from living in poverty, or living with a disorder like FAS or other cognitive impairments, would be the victims of the extension of mandatory sentencing laws (Robinson, 2013b). Should the laws be passed, a child as young as 11 could face a two-year sentence in prison.

In Canada, these problems may look familiar, including an overrepresentation of Aboriginal youth (Erickson & Butters, 2005; La Prairie, 2002; Yessine & Bonta, 2009), with Aboriginal youth receiving longer custodial sentences than non-Aboriginal youth (Latimer & Foss, 2005). This may be due to increased risk of poverty, unstable family homes, parental substance abuse, and negative peer associations (Yessine & Bonta, 2009). Adding to the confusion are poorly organized and run youth justice systems. In Ontario, there is no overall

strategy for addressing youth justice; instead the duties are overseen by three separate ministries, none of which is in charge of the system (McMurty & Curling, 2008). There is an overreliance on the justice system for minor, non-violent matters, which can lead to the youth seeing themselves as a future criminal, stigmatize the youth amongst his/her family and friends, restrict opportunities, and create the opportunity for criminal associations. These factors make future crime more likely rather than reducing the risk.

The long-term follow-up of young offenders has not been assessed formally. However, a recent study conducted in the United States of America examined the outcomes for youth placed in a juvenile detention facility (Aizer & Doyle, 2013). Using data gathered from the Chicago Public Schools, the Juvenile Court of Cook County, and the Illinois Department of Corrections, they analyzed the adult criminal record for 37,692 people who had been incarcerated as a youth. It was found that youth who had been incarcerated were significantly less likely to finish school than control participants as well as less likely to finish than other youth who had been arrested but not incarcerated. The authors also looked at the link between youth detention and adult incarceration. Adult incarceration was defined as presence in a correctional facility at any point by age 25. There was a strong relationship found between juvenile incarceration and adult incarceration; youth who had been detained were 22-26 percentage points more likely than children in the community to be incarcerated as an adult. This relationship holds when the severity of the crime is taken into account. It could be that a number of other factors (such as home life, parenting, racism, substance use, etc.) may make one more susceptible to crime as a juvenile and to follow a criminal lifestyle into adulthood; however, this does not negate the finding that incarceration does not appear to reduce criminal activity in adolescents and may increase the risk for future criminal activity. It also raises the question as to whether incarceration should be used in cases where public safety is not an issue.

1.2.2. Executive Function and Offending

To date, research on executive function in offending populations has been fraught with methodological problems, most significantly difficulty in defining what the term “executive function” encompasses (Herrero, Escorial, & Colom, 2010). Further, findings have been mixed as to the relationship between antisocial behaviour and executive function. Two types of offenders have received some individual attention in regard to this question: those who commit violent offenses and those who commit sexual offenses.

In a review on the neuropsychology of sexual deviance, Joyal, Black, and Dassylva (2007) noted that in general, the results of research support the theory that people charged with a sexual offense demonstrate frontal and/or temporal dysfunction. This is determined by their poorer performance on tasks of attention, inhibition, and verbal and non-verbal fluency (Gillespie & McKenzie; Kelly et al., 2002). However, Joyal and colleagues (2007) pointed out a number of methodological concerns with this research, such as the grouping of all sex offenders together and the absence of comparison groups.

Regarding violent offenders, Hancock, Tapscott, and Hoaken (2010) investigated the role of executive function in federal offenders incarcerated in a facility. Using 77 offenders, 65% of whom had served a sentence for violent crime, they used the *Delis-Kaplan Executive Function System* to determine executive function or dysfunction. It was determined that offenders who scored poorly on subtests related to impulsivity, concept formation, and cognitive flexibility were more likely to have committed a violent offense than offenders who performed to expectation. Zagar, Busch, Grove, Hughes, and Arbit (2009) found in their sample of 254 young offenders (127 with a charge of homicide, 127 with charges unrelated to violence) that youth who had committed homicide performed more poorly on the *Zagar Executive Function Checklist*. It should be noted that the checklist relies strictly on rater observation.

In general, the literature concerning executive function in offending populations is sparse and findings are mixed. It appears that there may be a relationship between deficits in executive function and antisocial behaviour, particularly for offenders with violent or sexual charges; however, more research is required in order to clarify the nature of the relationship, if one indeed exists.

1.2.3. Home Stability and Delinquency

There is debate in the literature as to the importance of a stable home life in protecting a youth from delinquent or criminal activity, regardless of prenatal exposure to alcohol.

Caldwell, Sturges, and Silver (2006) looked at the impact home and school environments have on the emotional and behavioural states of young offenders. Using a sample of 626 young offenders from Nevada, they used a number of subscales from the *Manifestation of Symptomatology Scale (MOSS)*, such as the Home Environment Scale to assess the behaviours and emotional states of the participants, the School Environment Scale to measure school-related attitudes and behaviours, the Compliance Scale to assess for one's likelihood of encountering

trouble with the law (it contains items related to one's propensity for getting in trouble and disregarding rules), and the Impulsivity Scale to identify a youth's control over impulsive, aggressive, and hostile behaviour. In addition, the *Massachusetts Youth Screening Instrument (MAYSI-2)* was used to assess for problems of depression, anxiety, anger, and irritability. They determined that in male juvenile delinquents, a supportive home environment was related to better compliance and lower scores on measures of impulsivity, which suggests fewer instances of acting out.

Van der Laan, Veenstra, Bogaerts, Verhulst, and Ormel, (2010) examined risk and protective factors for adolescent delinquency using a sample from a prospective Dutch cohort study that followed preadolescents to the age of 25, with the intention of better understanding their mental health and social development as they age. The sample for this particular study consisted of 2230 participants, and information was collected through parental interview and questionnaire. In addition, the youths completed the *Delinquency Scale* and teachers were asked to provide information on academic performance and the perceived social well-being of the youth in the classroom. They found that a supportive family environment was related to non-delinquency, whereas high rejection, dysfunctional family functioning, and high parental stress were found to be risk factors for delinquent behaviour.

Similar results have been found in an American longitudinal cohort study. Using a sample of 1389 participants taken from the National Longitudinal Survey of Youth (NLSY-79), Sullivan (2006) examined the relationship between delinquency and the family environment. Mothers were asked to complete the *Behaviour Problem Index (BPI)*, as well as provide information regarding the child's health, education, and social development. The youths completed a self-report measure querying substance use, delinquency, and peer associations. Sullivan discovered that the family environment had a protective effect on delinquency, as increases on the positive home environment measure corresponded to a decrease in the delinquency score. This finding suggests that the family is an extremely important factor in any treatment planning for youths with a myriad of problems.

It is possible that foster care may result in particular risk for children and youth with FASD, as foster parents are often unable or unwilling to provide the high levels of supervision required in raising a child with FASD (Paley & Auerbach, 2010). They may become frustrated when standard methods of behaviour control are ineffective. Multiple placement failures often

end up in group homes which are ill prepared to handle such challenging behaviour and where the child is exposed to peers who also have behaviour problems. Paley and Auerbach note that because of how such children are seen by the community, they are increasingly excluded from activities and groups which may moderate the chance of criminal involvement. In a 2008 report on the roots of violence in Ontario's youth, McMurty and Curling described a lack of adequate and accessible social and physical infrastructure as key contributing factors to youth violence. In addition, youth are at risk as they transition out of the foster care system, as they are often faced with issues such as lower education, unemployment, poverty, and criminal conviction (Paley & Auerbach, 2010).

The home environment is an important consideration for the proposed research, as children and youth with FASD have a higher incidence in foster care (Astley, 2006; Conry & Fast, 2000); in one sample of 61 participants with FASD, aged 12 to 40 years old, it was found that they had lived in an average of five principal homes in their lifetimes (Streissguth et al., 1991). Only nine percent remained with their biological parents. Some researchers estimate that 80% of children in foster care have been exposed prenatally to alcohol (Dicker & Gordon, 2004). Children in the child welfare system are more likely to have been exposed to ongoing parental substance abuse, parental psychopathology, domestic or community violence, neglect or abuse, placement disruptions, a number of caregivers, and the loss of important attachment figures (Paley & Auerbach, 2010). It is important to note that even in a sample compared to behaviourally challenged children, the majority of participants with FASD were not living in their biological home; alternatively, only one participant without FASD (but with significant behavioural issues) was living away from his/her biological family (Rasmussen et al., 2010). This indicates that youth with FASD are not more likely to be taken into care strictly because they exhibit behavioural issues; there are other variables that contribute to make home disruption more likely for children and youth with FASD.

Looking specifically at the relationship between home life and delinquency in youth prenatally exposed to alcohol, Lynch, Coles, Corley, and Falek (2003) examined home life stability as part of a larger project to determine risk factors for delinquency in prenatally exposed adolescents. They used a sample of 250 youths whose caregivers were recruited from an inner-city hospital, divided into three experimental groups: a control group unexposed to alcohol, a group of prenatally exposed and dysmorphic participants, and a group prenatally exposed but not

dysmorphic. The primary caregiver was asked to complete measures of parental warmth and involvement, the *Conflict Tactics Scale* (which assesses for exposure to violence and conflict within the family), the *Addictions Severity Index* to determine parental substance use, an index on negative peer influences, the *CBCL* to get at behaviour problems and the *Screening Survey of Children's Exposure to Community Violence*. The youth themselves were asked to complete measures of delinquent behaviour, substance use, and life stress. From their results, Lynch and colleagues determined that prenatal exposure to alcohol affected neither the frequency nor variety of self-reported delinquent behaviours, as there was no significant difference between groups. Instead, they found that family characteristics, such as low parental supervision, low parental warmth, and higher family verbal aggression contribute more significantly to delinquent behaviour.

In their study of delinquency in PAE-exposed adolescents, Schonfeld and colleagues (2005) also looked at the impact of home environment characteristics. Consistent with the findings of Lynch and colleagues (2003), they discovered home placement was predictive of delinquent behaviour, with higher rates of delinquency in alcohol-exposed youth in biological and foster homes, when compared with adoptive homes (Schonfeld et al., 2005). Of those participants who received a diagnosis of CD, four lived with their biological parents, three resided in foster care, and one lived in an adoptive home.

However, in a retrospective study examining 274 children in the welfare system who had experienced trauma, those children with FASD who had experienced trauma were noted by teachers and caregivers to demonstrate significantly more externalizing behaviours on the *CRS* than children not diagnosed with FASD who had also experienced trauma (Henry, Sloane, & Black-Pond, 2007). This finding suggests that the offending behaviour present in some individuals with FASD is not necessarily strictly a result of childhood trauma; instead, it may be that the CNS impairments present in FASD reduce the child's capacity to handle traumatic experiences and lead to maladaptive behaviour patterns (Brown et al., 2010).

1.2.4. Educational Opportunities and Delinquency

Education and achievement have also been found to play a role in delinquency and adolescent criminal behaviour. Early on in school (between Kindergarten and Grade 2), delinquent children evidence poorer achievement scores in reading, spelling, math, and handwriting than their non-delinquent counterparts (Katsiyannis, Ryan, Zhang, & Spann, 2008).

By middle school, the differences in achievement become even more pronounced. Low achievement rates have also been associated with an increased risk of reoffending, as research has discovered that a deficit in reading ability and a past history of special education resources were able to distinguish between recidivists and non-recidivists in a sample of male young offenders (Katsiyannis & Archwamety, 1997). In fact, access to educational resources has been found to be a moderating variable on criminal behaviour in youth (Streissguth, Barr, Koban, & Bookstein, 1996).

In addition, young offenders with experience in special education have scored significantly lower on measures of academic performance than offenders with no special education experience (Zabel & Nigro, 2001). In a sample of 130 young offenders in a juvenile detention facility, achievement scores were obtained through the *Tests of Adult Basic Education*. Participants with a history of receiving special education resources were compared to those without extra educational assistance. While both groups scored below their appropriate grade level compared to their non-delinquent peers, participants who had received special education resources in school scored significantly lower than those who did not on all areas of achievement: mathematics, language, and reading.

Similar results have been found in community samples of young offenders. Brown, Riley, Walrath, Leaf, and Valdez (2008) looked at achievement and school functioning in 157 non-incarcerated youth with justice system involvement. All participants were between twelve and seventeen years old, with a parent who had completed a routine intake assessment querying past educational problems (grade level, grade failure, drop out, etc.); parents were asked to complete the *Behaviour and Emotional Rating Scale – School Functioning Scale (BERS-SF)*, and the youth were required to fill in the *Child Health and Illness Profile – Adolescent Edition – Academic Performance Scale (CHIP-AE)* and the *Woodcock-Johnson Test of Achievement (WJ)*. Brown and colleagues found that achievement scores were low compared to age-appropriate norms, and similar to those found in incarcerated populations. Only 25% of their sample scored above the mean on the *WJ*, compared to 50% observed in non-delinquent community samples. Impairments were present across the achievement board, including applied problem solving, math fluency, passage comprehension, and reading fluency. Also akin to previous findings, youth who had previously received special education services performed significantly worse than their non-special education counterparts.

Li and Lerner (2011) examined the relationship between school engagement and delinquency, amongst other variables. Their sample was based on a longitudinal cohort study that consisting of following 1977 youths; the youths included in this study were involved in the longitudinal design between grades five and eight. Parents were asked to complete a standard demographics questionnaire; the youths completed measures on emotional and behavioural school engagement (modified form of the *Profiles of Student Life: Attitudes and Behaviours*), as well as delinquency (modified version of the *Monitoring the Future* questionnaire). They found that adolescents with higher rates of school engagement were less likely to engage in delinquent behaviour, whereas those with lower school engagement and poorer grades evidenced the highest rates of delinquency and substance use.

Interestingly, in their study of the effect of home and school environments on delinquency (described in greater detail above), Caldwell and colleagues (2007) found that a negative appraisal of, and experiences in, the school environment were more predictive of problems with impulsivity and compliance than negative home life experiences. As well, van der Laan and colleagues (2010) determined that a negatively perceived school environment in preadolescence increased the likelihood that a youth would become involved in serious delinquency at adolescent onset. They found that high academic achievement was the only school-related factor to reduce the likelihood of delinquency.

1.2.5. Substance Use and Delinquency

Substance abuse and use disorders are significantly more common amongst offending populations when compared to the general public. In a systematic review of the literature of substance use and abuse in offenders, Fazel, Bains, and Doll (2006) examined thirteen studies with a total sample of 7563 offenders. It was found that prevalence estimates for male prisoners range from 18-30% for alcohol abuse and 10-14% for drug abuse. Women prisoners had slightly lower estimates of alcohol abuse, ranging from 10-24%; however, estimates of drug abuse were higher for female offenders, ranging from 30-60%. The authors note that these are higher than rates found in the American general population, with male offenders two to ten times more likely to engage in substance abuse than men in the community, and the female offenders two to four times more likely to engage in alcohol abuse, and at minimum a thirteen fold increase in drug dependency (Kessler, McGonagle, Zhao, Nelson, Hughes, et al., 1994). Novak, McDermott, Scott, and Guillory (2007) looked at rates of substance abuse in a sample of offenders found Not

Guilty by Reason of Insanity (NGRI). Their sample consisted of 458 patients hospitalized after being found NGRI. Charts were coded on psychiatric diagnosis, including substance use disorders. A substance use disorder was diagnosed in 76 percent of all adult offenders.

It appears that sex offenders are more likely to be affected by substance abuse than non-sexual offenders. A meta-analysis of sex offenders and substance misuse and substance abuse disorders took into account 42 studies of substance abuse in sex offenders (Kraanen & Emmelkamp, 2011). It revealed that independent of the research method used, approximately half of the sex offending population under review was diagnosed with a lifetime substance abuse disorder. Between 25 and 50 percent of sex offenders had identified alcohol abuse, whereas rates of drug abuse were found in between 20 and 25 percent of sex offenders.

Violent offenders have been found to have significantly more serious alcohol treatment requirements than non-violent offenders. Bowes, Sutton, Jenkins, and McMurran (2009) looked at the treatment needs amongst a sample of 90 convicted and sentenced adult males detained in prison. In addition to answering questions about their index offense (for instance, was it classified as violent?), each participant was administered the *Alcohol Use Disorders Identification Test (AUDIT)*, a self-report screening measure of hazardous drinking. They found that 73% of the total prison population was in need of intervention to address their alcohol use. Participants incarcerated for violent crimes had more serious alcohol needs, as determined by higher scores on the *AUDIT*; violent offenders also reported more injuries due to their alcohol use than did non-violent offenders, suggesting that they engage in more risky behaviours that result in harm to themselves or others.

It appears that young offenders who were prenatally exposed to alcohol are at an increased risk of abusing substance, greater than that of young offenders without prenatal exposure to alcohol. Rates of substance abuse in young offenders with FASD are estimated to be as high as 83.8% (Conry & Lane, 2009; as cited in Fast & Conry, 2009) compared to estimates of 37.2% of the general youth offending population (Shelton, 2001). Rates of substance abuse in general in individuals with FASD are seemingly higher than for those without FASD. Denys, Rasmussen, and Henneveld (2011) found that 63% of their sample consisting of 24 non-offending participants who were either diagnosed with or suspected of having FASD reported past or present substance abuse. Another study found that prenatal exposure was a better predictor of substance use and abuse than familial alcohol use (Baer, Barr, Bookstein, Sampson,

& Streissguth, 1998). Using a sample of 464 families taken from the University of Washington Fetal Alcohol Follow-Up study, adolescent alcohol use was measured with the Lifestyle Choices Survey and a short version of the Rutgers Alcohol Problem Index, self-report instruments confirmed by parental report. Familial substance use was determined through parental responses on the Family Tree Questionnaire for Assessing Family History of Drinking/Drug Abuse Problems. Baer and colleagues found that prenatal alcohol exposure predicted alcohol problems in adolescents even when aspects of the postnatal environment (ex: measures of parenting style, current parental drinking) were held constant.

1.3. Summary

FASD are the result of prenatal exposure to alcohol and have associated growth and neurocognitive abnormalities, many of which are persistent and can lead to debilitating problems. While the diagnoses are gaining recognition throughout the medical and correctional community, many problems remain with assessment and diagnostic considerations. The disorders are difficult to identify as the presentation is often complex and overlapping with other disorders (Burd et al., 2003; Chudley et al., 2003; Nash et al., 2013). FASD are often comorbid with other disorders, both medical and psychological (Fryer et al., 2007; Doig et al., 2008). A maternal history of drinking is often difficult to reliably obtain (Astley, 2006; Burd et al., 2003a). Adding to the confusion have been the variable diagnostic criteria used by different clinics in different countries, which has led to inconsistent diagnoses. In response to this issue, Health Canada developed the Canadian guidelines for diagnosing FASD and these criteria have been adopted throughout the nation; it appears that as of the writing of this document, Canada was the only nation to have nationally accepted standards for diagnosis (Chudley et al., 2005).

As part of the diagnostic process, it is important that an individual with suspected FASD undergo a thorough neuropsychological examination. People with FASD have been found to have impairments in a number of areas: brain structure and neurological deficits (Astley et al., 2009; Coles et al., 2011; Franklin et al., 2008; Green et al., 2009; Jacobsen et al., 2008; Kalberg et al., 2006; Nardelli et al., 2011; O'Brien et al., 2012; Stephen et al., 2012), cognition (Carr et al., 2010; Chasnoff et al., 2010; Howell et al., 2006; McGee et al., 2009; Nash et al., 2013), receptive and expressive language (McGee et al., 2009; Nash et al., 2013), achievement in school (Howell et al., 2006; Olson et al., 1998; Streissguth et al., 2004), memory (Kully-Martens et al., 2012; Manji et al., 2009; Mattson & Roebuck, 2002; Olson et al., 1998; Pei et al., 2013),

executive functioning and abstract reasoning (Brown et al., 2010; Green et al., 2009; Kully-Martens et al., 2013; Rasmussen et al., 2006; Schonfeld et al., 2006), attention (Connor et al., 1999; Green et al., 2009; Mattson et al., 2006), and adaptive behaviour and social skills (Greenbaum et al., 2009; Jirikowic et al., 2008; McGee et al., 2009; Nash et al., 2013; Rasmussen et al., 2010; Schonfeld et al., 2006; Streissguth et al., 2004). These are considered the primary disabilities of FASD.

Secondary disabilities in FASD are those difficulties that are thought to arise from the mismanagement (or inability to manage) the primary deficits, and include school disruptions, substance use, and involvement with the criminal justice system. There has been very little empirical research on FASD in the justice system, but researchers hypothesize that the greater risk of criminal involvement in those with FASD is tied to primary deficits, such as their vulnerability for manipulation, coercion, and false confession due to social skills impairment (Keil et al., 2010), impaired reasoning (Burd et al., 2010), trust of authority figures (Abel, 1998), and inability to generalize from past mistakes and make cause-effect connections (Boland et al., 1998). Other specialists believe that criminal activity is directly tied to the primary deficits, such as impaired planning and organization due to difficulties with executive functions (Fast et al., 1999), verbal deficiencies (Akbarian, 1992; Brown et al., 2010), or problems with attention (Green et al., 2009) or memory (Manji et al., 2009) that impede one's capacity to learn from experience. As empirical research on this connection has not yet been conducted, such hypotheses are theoretical at this point. The few studies that have examined the rate of FASD in offending populations indicate that such individuals are overrepresented in delinquent (Disney et al., 2008; Schonfeld et al., 2005) and correctional populations (Fast et al., 1999; Popova et al., 2011).

Professionals who work with offenders with FASD have begun to recognize the role the disorder plays in contributing to criminal activity, and recommend that accommodations be made in the justice system for people with FASD, as the current practice of treating them the same as other offenders is not garnering results (CBA, 2010; Lafond, 2001). However, people with FASD are also likely to be overrepresented in other populations known to be at an increased risk for future criminal behaviour. For example, rates of FASD in foster care placements is disproportionately high (Astley, 2006; Conry & Fast, 2000; Dicker & Gordon, 2004) and a disruptive home life has been found to be linked to delinquency and criminal behaviour

(Caldwell et al., 2006; van der Laan et al., 2010). People with FASD are at a higher risk for developing a substance use or abuse disorder (Baer et al., 1998; Denys et al., 2011), which also results in a greater risk for criminal activity (Fazel et al., 2006; Novak et al., 2007).

The increased likelihood of delinquency and criminal involvement for individuals with FASD is well established (Fast et al., 1999; Schonfeld et al., 2005; Streissguth et al., 2004); less clear are the precise mechanisms underlying this association. There is debate as to whether it is the organic brain damage resulting from prenatal exposure to alcohol that is the root cause for the heightened risk, or the deficient and abusive households many prenatally exposed children experience. Paley and Auerbach (2010) suggest that a significant number of prenatally exposed children and youth will be faced with both prenatal and postnatal adversities. The current research was conceived to be of assistance to individuals with FASD and the criminal justice system by identifying the neurocognitive and psychosocial differences between young offenders with and without FASD. Interventions can then be more appropriately targeted for offenders with FASD to minimize the risk of recidivism. More importantly, this research serves as another piece of the puzzle in elucidating the relationship between FASD and criminal behaviour, which may ultimately lead to preventative efforts.

2. HYPOTHESES

2.1. Hypothesis 1: Neurocognitive Profile

2.1.1. Hypothesis 1a

Youth with FASD will have a different pattern of deficits on neuropsychological assessment from youth without FASD.

2.1.2. Hypothesis 1b

Youth with FASD will demonstrate a different pattern of deficits on the neuropsychological assessment, depending on their specific diagnosis. That is, I expect youth with a diagnosis of FAS to evidence more impairments than those with a diagnosis of pFAS or ARND.

2.2. Hypothesis 2: Neurocognitive Scores and Offending

It is predicted that the crimes the participants have accrued will be related to the neuropsychological impairments found in the assessment.

2.2.1. Hypothesis 2a

It is expected that participants with evidence of memory impairment will have accrued system generated charges; that is, charges related to breaches and failure to comply, as compliance with conditions requires the offender to remember which restrictions he is under. As such, remembering one's conditions is a real-life verbal memory task.

2.2.2. Hypothesis 2b

It is predicted that having generated a sexual offense charge will be associated with deficits in executive functioning.

2.2.3. Hypothesis 2c

It is predicted that having generated a violent offense charge will be associated with deficits in executive functioning.

2.2.4. Hypothesis 2d

It is expected that if criminal activity in individuals with FASD is predominantly due to the organic brain damage caused by prenatal exposure to alcohol, as I am hypothesizing, the relationship between neurocognitive scores and criminal charges will be stronger in the group of offenders with FASD. All scores will be reported in correlation coefficient form for each group.

2.3. Hypothesis 3: Home Stability and Offending

It is anticipated that there will be a relationship between criminal activity and the participant's environmental stability.

2.3.1. Hypothesis 3a

It is predicted that long term stability in living arrangements as evidenced by living consistently in one setting will be related to fewer crimes.

2.3.1.1. Hypothesis 3.a.1. Youth who have had greater environmental stability will have accumulated fewer charges than those whose home environments have been disrupted between birth and age seven.

2.3.1.2. Hypothesis 3.a.2. Youth who have had greater environmental stability will have accumulated fewer charges than those whose home environments have been disrupted between ages eight and twelve.

2.3.1.3. Hypothesis 3.a.3. Youth who have had greater environmental stability will have accumulated fewer charges than those whose home environments have been disrupted between ages thirteen and eighteen (or their age at the time of the assessment).

2.3.2. Hypothesis 3b

Because of the high needs individuals with FASD present, it is anticipated that young offenders with FASD will benefit more from home stability. Thus, it is expected that the relationship between criminal activity and home instability will be stronger for offenders with FASD, when compared to the sample without a diagnosis.

2.4. Hypothesis 4: Educational Assistance and Offending

It is predicted that educational resources will be related to neuropsychological impairments and criminal activity.

2.4.1. Hypothesis 4a

Access to special educational resources (special educational placement, resource room time, etc.) will be significantly related to more neuropsychological impairments, as one could expect those who qualify for special services to demonstrate neurological impairments in some capacity.

2.4.2. Hypothesis 4b

It is expected that neurological deficiencies left unaddressed will lead to more criminal offenses. As educational assistance has been found to moderate criminal behaviour, I predict

those offenders who were provided with educational resources will have committed fewer offences at a level that reaches statistical significance.

2.5. Hypothesis 5: Substance Use and Offending

It is expected that current alcohol and drug use will be associated with more charges.

2.5.1. Hypothesis 5a

Participants who have used alcohol will have significantly more charges than those who have not.

2.5.2. Hypothesis 5b

Participants who have used marijuana will have accrued more charges than those who have not.

2.5.3. Hypothesis 5c

Participants who have used other drugs will have accrued more charges than those who have not. Substances that would fall under this classification would include methamphetamines, LSD, psilocybin mushrooms, heroin, cocaine, ecstasy, prescription medications, etc. These drugs are designated as “other” because while some of the participants will have tried the aforementioned substances, this kind of drug use is much less common than alcohol or marijuana use. As such, the data will be aggregated to maintain statistical power.

2.5.4. Hypothesis 5d

I expect the relationship between substance abuse and crime will be significantly stronger for offenders with FASD.

3. METHOD

3.1. *Participants*

The sample for this project consisted of young offenders, ages 12 to 18. Participants were referred to Dr. Nanson by the court for assessment. Dr. Nanson received 202 referrals from the youth court; of these, 197 were eligible to participate in this study. Four youths were referred but did not attend the assessment and one individual refused consent for her data to be included in the database, as indicated by the fact that the section of the consent form addressing the database was crossed out and initialed by the examinee. All participants were referred through the youth court and so were charged at age 17 or younger; however, by the time the assessment was conducted, four participants had reached the age of maturity. The mean age of the sample was 15 years old ($SD = 1.41$). The sample was 77.2% male. The youths were predominantly First Nations, making up 79.2% of the sample; 13.2% of the sample was Caucasian and 7.6% were classified as other ethnicities, such as African, Asian, and Hispanic.

It is important to note that this sample is not representative of the general youth offending population. The referred youth were a largely heterogeneous group of young offenders. Referral criteria were unspecified; the only requirement for assessment was that one member of the youth's treatment team (youth worker, social worker, judge, lawyer, etc) had questions about the youth's neurocognitive functioning, possibly due to a history of significant managerial problems, past record of head injury, evidence of prenatal alcohol exposure, some indication of a cognitive delay, or a combination of the aforementioned factors. All participants were screened for FASD at the outset of referral; each youth was administered the same neurocognitive battery but only those who met criteria for possible FASD (i.e., evidence of prenatal alcohol exposure) were referred to a medical specialist for diagnosis. Thus the control group is not homogeneous or representative of young offenders in general and is, in many ways, indistinguishable from the FASD sample. This study does not provide information as to the differences between young offenders with FASD and the majority of young offenders, who do not present with serious management issues. It does highlight if the deficits and social situations experienced by youths with FASD can distinguish them from other groups of challenging and impaired offenders, as well as provide information on the offending patterns of this sample of highly at-risk youth. In addition, it is important to note that youth included in the control sample may have been exposed

to alcohol in utero but did not meet criteria for a diagnosis of FASD. Thus, this research cannot inform as to differences between prenatally exposed young offenders and those without exposure; it can only identify differences between youth diagnosed with FASD and those who do not qualify for a diagnosis. It is also of use to note that the majority of research involving young offenders will include some youth who have been prenatally exposed to alcohol and/or have FASD but are embedded within homogenous groups.

3.2. Materials

Because the sample was collected over a twelve-year interval, the battery of tests given to the youth changed as newer, more appropriate measures became available. For example, when the *Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV)* became available, it was substituted in place of the *Wechsler Intelligence Scale for Children, Third Edition (WISC-III)*. In order to allow for interpretation of scores from different tests, standard deviation scores (z scores) were used for analysis. The procedures for each domain are discussed below.

The neuropsychological assessment consisted of assessment of each of the psychological domains proposed in the Canadian guidelines: achievement, cognition, executive functioning, memory, language abilities, and attention. Each of the domains is described in greater detail below, as outlined in the operational definition and measures used to assess each area. These Canadian guidelines require that the affected individual be considered to have central nervous system (CNS) deficits if their test scores fall more than two standard deviations below the mean on three domains, or if they have discrepancies within domains of more than one standard deviation (e.g. verbal versus visual memory).

When considering the appropriateness of psychological instruments, there are two important statistical concepts to consider. The first is reliability, which is the consistency of a test's results over time and across changing conditions (Field, 2009). Reliability is measured by a coefficient, ranging from .00 (no reliability) to 1.00 (perfect reliability; Sattler, 2008). Murphy and Davidshofer (2005) have described any coefficient under .59 as having very low or very poor reliability; coefficients between .60 and .69 have low or poor reliability; those between .70 and .79 are considered to have moderate or fair reliability; coefficients falling between .80 and .89 are described as having moderately high or good reliability, and any coefficient over .90 has high or excellent reliability.

There are four central types of reliability. Internal consistency is determined from a single administration, and assesses the homogeneity of items throughout the measure (Sattler, 2008). Test-retest reliability is an estimate of instrument stability over short intervals, during which one would not expect an examinee's scores to change. Alternate forms reliability coefficients result from administering two different yet parallel test forms to the same sample; reliability is determined by examining the extent of the agreement between scores on each measure. Finally, interrater reliability represents the extent to which different raters agree when scoring the same instrument.

The second important concept in evaluating assessment measures is validity. Validity is the extent to which a test measures what it is purported to measure (Field, 2009). This ties in directly to our ability to draw accurate conclusions based on the instrument results (Sattler, 2008). As with reliability, there are a number of different forms of validity. A test has content validity when the items of the measure represent the domain the instrument claims to assess. Consideration of content validity must take into account the appropriateness and type of items, the breadth of the item sample, and the fashion in which the content of the domain is assessed by the items. Face validity refers to the perception of examinees and examiners in taking the test; does the measure *appear* to be a reasonable measure of the domain of interest?

Construct validity is based on the degree to which an instrument measures a particular psychological construct (for example, intelligence or memory; Sattler, 2008). It is comprised of two parts: convergent and discriminant validity. A test has convergent validity when measures of the same construct with different format (i.e., essay, multiple choice, etc) correlate well with each other. Discriminant validity represents the extent to which instruments designed to assess different domains *do not* correlate with each other, indicating that they are genuinely assessing different constructs.

Criterion-related validity refers to a relationship between the test results and some outcome, or criterion (Sattler, 2008). Examples of common criteria are test scores on other measures intended to assess the same domain, or independent ratings or classifications. There are also two forms of criterion-related validity. Predictive validity is based on the correlation between the results of a measure and the results of a criterion at a later date (for example, a reading aptitude test at the beginning of Grade Three and a reading test at the end of the year). Concurrent validity refers to the correlation of the measure of interest with other related

instruments. For instance, as reading ability is linked to phonetic skills, we could expect tests of reading and tests of phonetic ability to correlate. Criterion-related validity estimates are typically expressed as a correlation coefficient.

Finally, a brief discussion of effect size is warranted, so that one may be able to understand the magnitude of an effect instead of merely whether a relationship is significant or not. One of the most common indicators of effect size is Cohen's *d*; its ubiquity makes it possible to compare an increasingly large number of scientific studies (Thalheimer & Cook, 2002). According to Cohen's *d*, an effect size of .20 is small, of .50 is medium, and of .80 is large.

With these core concepts in mind, it is more possible to adequately evaluate the instruments themselves.

3.2.1. Achievement

The first domain assessed following the Canadian guidelines is academic achievement; that is, the acquisition of basic skills in reading and mathematics. For the purpose of FASD assessments, achievement was measured by the appropriate version of the *Wide Range Achievement Test (WRAT-3 or WRAT-4)*.

The *WRAT-3* was developed to assess reading, spelling, and arithmetic skills (Parker, 2002). Subtests involve simple word reading, spelling words, and written math computation problems; each subtest begins with easier items and progressively becomes more difficult. As it is briefer than most achievement batteries, it can be used as a screening measure, or an initial evaluation of a person's abilities. It can also be used as part of a larger battery to obtain a comprehensive understanding of one's academic skills. The *WRAT-4* added a subtest to assess reading comprehension abilities, in addition to simple word reading (Wilkinson & Robertson, 2006). This subtest requires an individual to fill in the word missing from a sentence. Both editions of the test provide an alternate form (blue and tan for the *WRAT-3*; blue and green for the *WRAT-4*), which can be used interchangeably or for comparison purposes. Scores can be converted to standard scores, percentile ranks, stanines, normal curve equivalents, and/or Rasch ability scores. Grade based norms are available only for the *WRAT-4*. Overall, the measures are quick, easy to administer, and psychometrically sound, as is discussed below.

The *WRAT-4* content on the Reading and Math Computation subtests are largely unchanged from the *WRAT-3* (Wilkinson & Robertson, 2006). The biggest change between editions is in the addition of Sentence Comprehension as a subtest. While reading comprehension

is no doubt an important part of achievement, it is not possible to compare scores for the earlier participants as their testing did not generate a reading comprehension score. However, following the central analyses, tests can be run on the subset of participants who were administered the *WRAT-4* to detect for differences related to comprehension of written material.

Both the *WRAT-3* and the *WRAT-4* were normed on large samples (over 3000 participants), matched as closely as possible to the general United States population in age, gender, race/ethnicity, and regional residence (Strauss, Sherman, & Spreen, 2006). In addition, Canadian norms are available. The *WRAT-4* sample was additionally matched on educational attainment (Wilkinson & Robertson, 2006). On the *WRAT-3*, median reliability estimates of internal consistency are high to very high (.86-.91; Strauss et al., 2006). It has also demonstrated very strong stability, with test-retest coefficients ranging from .91-.98. Data on validity was more difficult to ascertain, although the authors assert that the *WRAT-3* underwent numerous statistical analyses (Wilkinson & Robertson, 2006). The *WRAT-3* correlates moderately with IQ scores, as determined by the *Wechsler Adult Intelligence Scale – Revised (WAIS-R)* and *WISC-III*, and it shows moderate to high correlations with other measures of achievement, such as the *California Achievement Test* and the *Stanford Achievement Test* (Strauss et al., 2006).

The *WRAT-4* similarly evidences good internal reliability, with the subtest's mean alpha coefficients ranging from .83 to .93 (Wilkinson & Robertson, 2006). Regarding validity, 77% of the test material in the *WRAT-4* is kept over from the *WRAT-3*; items were eliminated only after extensive content review by external experts, who were instructed to analyze the items for their current relevance and any ethnic or gender bias. The *WRAT-4* was found to correlate significantly with the *Wide Range of Achievement Test, Expanded Edition (WRAT-Expanded)*, with coefficients ranging from .63 to .80; math subtest scores were found to correlate more with other math subtests than with reading subtest, and vice versa. The Word Reading subtest has been shown to correlate moderately to moderately high with other measures of word recognition in achievement testing (.71 with the *WIAT-II* Word Reading; .66 with another widely used achievement test, the *Woodcock Johnson III [WJ III]* Basic Reading; .76 with the *Kaufman Test of Educational Achievement: Second Edition [KTEA-II]* Comprehensive Letter/Word Recognition). Similarly, sentence comprehension correlated moderately with the *WIAT-II* Reading Comprehension subtest (.61), the *WJ-III* Reading Comprehension subtest (.60), and the *KTEA-II* subtest for Reading Comprehension (.40).

There are significant correlations between the Math Computation subtest on the *WRAT-4* and *WIAT-II* Number Operations (.92), *WJ-III* Mathematics Calculation (.64), and the *KTEA-II* Math Computation (.75 on the comprehensive form). The *WRAT-4* has demonstrated the ability to significantly distinguish individuals with a learning disorder from low and high cognitive functioning participants (Wilkinson & Robertson, 2006).

3.2.2. Cognition

The second domain assessed within the Canadian guidelines is cognition. Cognition is what we consider to be intellectual functioning, or basic intelligence; it is often represented by an IQ score. This domain was operationally defined as that which is measured by the age appropriate Wechsler Scale, (*WAIS-III* or *WAIS-IV* for youth aged 16-18 or the *WISC-III* or *WISC-IV* for youth aged 12-16). Each scale consists of ten tasks and provides scores for verbal comprehension, perceptual reasoning, working memory, and processing speed, as well as a general (or “full-scale”) score to represent overall functioning. Verbal comprehension consists of general verbal ability, capacity to comprehend verbal material, and verbal knowledge (Wechsler, 2008). Perceptual reasoning skills are representative of fluid reasoning, spatial processing, visual-motor integration, and attention to detail. Processing speed is an estimate of attention, motor coordination, planning, and visual scanning. Working memory represents one’s ability to hold information in short-term memory and manipulate it to achieve some end.

Subtests for each edition of the *WAIS* require one to recognize patterns (using blocks to build a design, or choosing a missing element from a pattern), define vocabulary, draw associations between two objects, repeat numbers, perform oral math problems without the use of scrap paper, copy designs, visually scan distracter objects for a target design, and answer questions related to general knowledge, among other things. Each edition provides four supplementary subtests that can be substituted in to complete a domain score, should one of the core tests be rendered ineligible for some reason (an error in administration, for example). Some subtests were changed from one scale to another. For example, the third edition contained a subtest related to general comprehension questions (how to behave in certain situations, does the individual understand the purpose of government regulation or the free press, etc); in the fourth edition, this subtest is a supplementary test to be used if one of the core tests is ineligible. Other subtests were discarded entirely, such as a task asking people to arrange a set of cards in order to tell a story. These subtests were replaced in the fourth edition with new tasks, such as choosing

from three options to complete a picture puzzle. However, the majority of subtests between the two versions of the test remained the same, although norms and specific items on subtests were updated.

The *Wechsler Adult Intelligent Scale*, versions Three and Four (*WAIS-III*; *WAIS-IV*) were normed on large samples chosen to represent the general population in terms of age, gender, education and ethnicity (Strauss et al., 2006); in addition, Canadian norms reflective of our unique societal milieu are available. Reliability coefficients for internal and test-retest reliability are high for the most part, with the full-scale score demonstrating the most consistency, followed by verbal subtests; the non-verbal subtests evidence the greatest variability. Practice effects can be a concern; however, if the youth referred to us had extensive cognitive testing completed recently prior to his referral, Dr. Nanson would request the administrator send the raw data to evaluate, as opposed to retesting the youth. In addition, the *WAIS* correlates substantially with other measures of intelligence, moderately with achievement and memory tests, and moderately high with years of education (Strauss et al., 2006).

Participants under the age of 16 were administered the *Wechsler Intelligence Scale for Children*, either the Third or Fourth Edition (*WISC-III*; *WISC-IV*). These scales are very similar to the adult versions of the test, with age appropriate material and norms. Scores are similarly provided for verbal comprehension, perceptual reasoning, working memory, and processing speed. Children and youth are asked to draw associations between two items, define words, create designs with blocks, repeat numbers back, copy designs, visually scan for a target design amongst a group of distracters, define words, and answer questions related to comprehending the world around them. Larger changes were made between the *WISC-III* and the *WISC-IV* than between the *WAIS-III* and *WAIS-IV*; ten of the subtests were retained and revised, three subtests were deleted (or relegated to supplementary status), and five more added (Smith, 2003). Test makers decreased the focus on knowledge learned in school and attempted to get at more basic abilities by added subtests related to pattern recognition and working memory skills. Despite these changes, the two versions of the test correlate significantly, and have a minimum acceptable reliability and validity statistics.

Both versions of the test are said to have excellent reliability, with internal consistency reliability coefficients for each domain ranging from .81 to .97 (Sattler, 2008). Estimates of test-retest reliability range from .79 to .89, with the Full Scale IQ providing the greatest stability of

results (Wechsler, 2003). The validity of the *WISC-IV* was predicated on the validity of the *WISC-III*, as much of the test is the same as its predecessor; validity studies on the *WISC-III* indicate that it has acceptable construct, concurrent, and predictive validity (Sattler, 2008). In addition, the *WISC-IV* was found to significantly correlate with the *WISC-III*, other Wechsler measures of intelligence (such as the *Wechsler Abbreviated Scale of Intelligence* [*WASI*] and the *Wechsler Preschool and Primary Scale of Intelligence, Third Edition* [*WPPSI-III*]), and measures of achievement, memory, emotional intelligence, and adaptive behaviour, indicating that the *WISC-IV* has good criterion validity. Studies listed in the Technical and Interpretive Manual (Wechsler, 2003) and factor analysis (Sattler, 2008) suggest that the *WISC-IV* has good construct validity, as it is a good representation of general intelligence.

3.2.3. Executive functioning

The next domain of the Canadian Guidelines assessed was executive functioning, which involves complex processes such as organization, planning, abstract thinking, rule acquisition, and response inhibition. Executive functioning was operationally defined as that measured by select subtests of the *Delis-Kaplan Executive Functioning System (D-KEFS)*, the *Controlled Oral Word Association Test (COWAT)*, and the *Wisconsin Card Sorting Task (WCST)*.

The *D-KEFS* is a battery consisting of nine subtests to assess a variety of executive functions. The Trail Making Test is a measure of cognitive flexibility and visual-motor skills that requires individuals to scan a page and identify and eliminate a target number, connect numbers and/or letters in a sequential order, or follow a line as a measure of motor capacity (Delis, Kaplan, & Kramer, 2001). The Verbal Fluency subtest asks an individual to list as many words as one can for the letters F, A, and S within a sixty second timeframe. This assesses one's capacity to fluently produce verbal information. The design fluency task requires one to generate original designs under a set of guidelines and measures fluent productivity in spatial reasoning. The Colour-Word Inference Test is much like the Stroop test, in which individuals are asked to scan a list of colour words and say not the name of the word but the colour of the ink in which it is printed (for example, GREEN may be printed in blue ink, in which case one would be asked to say "blue"). This is a measure of verbal inhibition. The Tower Test requests that participants construct a tower out of a series of rings while following a set of instructions; this test is an assessment of impulsivity, planning, and reasoning. The 20 Questions Test is one in which the participant asks the examiner questions; the examiner had chosen an object on a page of 30

pictures, and the test taker must identify which object was chosen in the fewest number of questions possible. This subtest measures hypothesis testing, and spatial and verbal abstract thinking. The Word Context Test is an evaluation of deductive reasoning, for which individuals are provided a mystery word for which they must decipher the meaning from clue sentences. The Sorting Test was based on the *California Card Sorting Test* (Delis, Kramer, Kaplan, & Holdnack, 2004), and is a measure of problems solving, cognitive flexibility, and verbal and spatial concept formation (Delis et al., 2001). Finally, the Proverbs Test, for which individuals are asked to provide explanations for regular and irregular proverbs, is a measure of metaphorical thinking and abstract thought; responses are scored based on accuracy and abstractness.

Not all subtests were administered to all participants, and none received the Word Context subtest as it was not included in the battery. Norms for the Proverbs Test are only available for those over the age of sixteen (Delis et al., 2001), so younger participants were not administered this subtest. In addition, some participants were unable to grasp the concept behind some of the more complex tasks, such as the 20 Questions Test, and so these subtests were discontinued to maintain rapport and keep the participant from becoming overly frustrated.

The *D-KEFS* norming group consisted of 1750 individuals between the ages of eight and eighty-nine (Strauss, Sherman, and Spleen, 2008). The sample was chosen so as to best represent the demographic make-up of the United States of America in the 2000 census data. Estimates of stability demonstrate low to high test-retest correlations across the nine subtests (Davis, 2010). Internal reliability coefficients range from .43 to .84 across the age groups and each task. Research regarding the validity of the *D-KEFS* is variable. One consistent finding is a high relationship between the Sorting Test and other measures of executive functioning. There is evidence of convergent validity for the measure, as it was found to moderately correlate with the *WJ-III*. In addition, some research has found the *D-KEFS* to effectively distinguish between children with epilepsy and normal controls and was highly correlated with parental ratings of executive functioning (Parrish, Geary, Jones, & Seth, 2007), indicating ecological validity.

For each participant, we have data on verbal fluency, which is the capacity one has to simultaneously generate lexical responses fluently while abiding by several restrictions (Delis, Kaplan, & Kramer, 2001). The earlier tested participants were administered the *COWAT*. This task is exactly the same as the Letter Fluency subtest of the *D-KEFS*, although alternative forms

include the letters CFL and PRW as stimuli (Ruff, Light, & Parker, 1996). Responses made within a 60 second window are recorded and scored (Lezak, 1995). The *COWAT* was normed on a sample of 360 participants aged 16 to 17 years old, and care was made to ensure heterogeneity of the group in regard to age and education (Ruff et al., 1996). Tests of internal validity reveal an alpha coefficient of .83, indicating internal validity is acceptably high. Test-retest estimates of stability are moderately high, at .74, although Ruff and colleagues warn of the possibility of small practice effects upon retaking the test.

The Letter Fluency subtest of the *D-KEFS* was found to have high to very high internal consistency and test-retest reliability, with correlation coefficients over .80. Less evidence is available for validity on the Letter Fluency subtest, although it has correctly distinguished between healthy controls and people with frontal lobe lesions (Baldo, Delis, Wilkins, & Shimamura, 2001), and between people who were or were not exposed to alcohol prenatally (Schonfeld, Mattson, Lang, Delis, & Riley, 2001).

The majority of participants were administered the *Wisconsin Card Sorting Test (WCST)* in place of the *D-KEFS*. This measure was created to assess abstract reasoning and ability to shift cognitive strategies in light of changing stimuli (Clark & Roman, 2006). It evaluates strategic planning, organized searching, and the incorporation of feedback to change strategies where required. Using a set of four stimulus cards and 64 response cards that consist of four shapes, four colours, and four numbers, individuals are asked to determine the accurate sorting principle (i.e., by form, colour, or number) and keep the rule set across changing conditions (Davis, 2010). Feedback on whether the test taker is correct or incorrect is provided, and the individual is expected to incorporate the feedback into their decision making process. The test provides 13 different scores, only three of which have been determined to be clinically useful: the number of categories completed, perseverative responses, and perseverative errors.

The *WCST* was normed on a sample of 899 healthy participants between six and ninety years old (Clark & Roman, 2006). The adult sample was stratified to best correspond to U.S. census data, but the child and adolescent sample was not (Davis, 2010). Studies of interrater reliability reveal coefficients ranging from .88 to .93. Estimates of intrarater reliability reveal coefficients ranging from .91 to .96 (Clark & Roman, 2006). Research on the validity of the *WCST* provides evidence that the measure is valuable in discerning between healthy and impaired executive functioning in a variety of neurological and psychological problems, such as

closed head injuries, seizure disorders, schizophrenia, and learning disabilities in children. Overall, extensive research indicates the *WCST* is a valid assessment of executive functioning in adults; validity findings in youth and children have been more mixed (Davis, 2010). Unfortunately, studies comparing the *WCST* to the *D-KEFS* have largely revealed that the two instruments do not correlate well, as is documented in Table 1. It appears that the strongest correlations can be found between the Number of Categories completed on the *WCST*, and the number of Correct Free Sorts on the Sorting test of the *D-KEFS*. These scores may potentially be comparable, but the relationship between the two instruments is not strong. The *D-KEFS* was added to the battery relatively late in the proceedings, and only 19 participants were administered the *D-KEFS* in place of the *WCST*; thus it was decided that analyses for executive functioning should focus on those subtests of the *D-KEFS* with corresponding measures in the previous test battery, for the sake of consistency. As most of the participants were administered the *WCST*, the number of perseverative errors made on the *WCST* was included as an indication of inhibition.

3.2.4. Attention

The next domain within the Canadian guideline is attention. Attention is also a complex structure that consists of concentration, sustained focus, and processing speed. Impairments in attention were operationally defined as deficient performance on the *Trail Making Test (TMT)* A and/or B, which correspond directly to Trails Conditions 2 and 4 of the *D-KEFS*.

The *TMT* is an easy to administer paper and pencil measure of complex visual scanning, and is given in two parts (A and B; Lezak, 1995). The *TMT-A* asks the individual to connect a series of numbered dots following numerical order. The *TMT-B* requires the test-taker to connect a series of numbered and lettered dots, following numerical and alphabetical order, alternating between numbers and letters. The time taken to complete each task is compared to age appropriate norms to determine impairment. In constructing the *D-KEFS*, Delis and colleagues (2001) adopted these exact measures and incorporated them into the Trails subtest, as Condition 2 and Condition 4, respectively. Thus, the same measure was possible for all participants, regardless of when they received the battery. One possible confounding variable is that the earlier participants would have only received the two conditions of the *TMT*, whereas those who completed the *D-KEFS* would have completed three more visual scanning conditions around the two of interest, possibly leading to better understanding of the instructions. Alternatively, participants who were administered the *D-KEFS* may have been more cognitively tired due to the

Table 1: Review of Findings Regarding the Relationship Between the *D-KEFS* and the *WCST*

Name of paper	Subjects	Purpose	Results	Relevance to our work
Razani et al., 2007	35 pts with dementia; 35 age matched normal controls	Comparison of executive functioning scores and activities of daily living	No differences between dementia pts and normal controls on <i>MMSE</i> (i.e. very mild dementia or some of their “normal” controls were also dementing). Used spouses so this is possible. <i>DKEFS</i> sorting, FAS, <i>WCST</i> errors differentiated the groups.	Very different age groups, and types of neurological disease. We have FAS data. They didn’t use <i>DKEFS</i> verbal fluency measure. Could argue that <i>DKEFS</i> sort and <i>WCST</i> errors are tapping similar functions.
Fisher, 2007	28; aged 8-12; advanced chess players vs. less advanced chess players	Examine differences in executive functioning and intelligence; look at relationship between	Small to moderate (.4-.6) correlations between <i>D-KEFS</i> subtests and other cognitive constructs (ex: IQ). High-performing chess players scored	Younger age group; no brain dysfunction. Little significant relationship between <i>D-KEFS</i> and measures of executive functioning; no

		<i>D-KEFS</i> subtests and other measures of cognitive functioning	higher on Trails, Sorting, Word Context, symbolic working memory, visual IQ, and FSIQ.	correlations approaching significance for the <i>WCST</i> and <i>DKEFS</i> subtests.
Beatty et al., 1994	21 pts with schizophrenia or schizoaffective disorder (SCZ); 28 age and education matched normal controls	Compare performance on the <i>WCST</i> with the <i>CCST</i>	SCZ pts performed more poorly on global mental status exam, most measures of problem solving as measures by <i>WCST</i> . On <i>CCST</i> , controls and SCZ performed similarly on total sorts; absolute number and percentage of correct sorts lower for controls. SCZ fewer points for verbal explanations, more perseverative verbal responses, poorer performance in cued-sorting phase.	Different age groups; different neurological dysfunction. Measures of concept attainment from <i>CCST</i> positively correlated with measures of concept attainment from <i>WCST</i> ; negatively correlated with perseverative errors on the <i>WCST</i> . <i>CCST</i> Correct Free Sorts significantly correlated with <i>WCST</i> Number of Categories (.64) <i>CCST</i> Perservative Free Sorts significantly correlated with <i>WCST</i> NOC (.72)
Beatty & Monson, 1990	27 pts with Parkinson's Disease (PD), aged	Clarify nature of problem solving impairment in PD;	PD pts scored lower on the SECIMS (screening tool for cognitive impairment).	Very different age group, different neurological impairment. Overall pattern of results for the

		50-80; 25 age and education matched normal controls	compare performance on <i>WCST</i> and <i>CCST</i> re: sensitivity to global and specific deficits	<p>No significant difference between groups on letter fluency or <i>WCST</i>.</p> <p>On <i>CCST</i>, PD pts made fewer correct sorts, attempted fewer sorts, more perseverative errors than controls.</p> <p>PD pts scored lower for verbal explanations</p>	<i>WCST</i> and <i>CCST</i> similar, but no significant correlations between subtest scores.
8	Greve et al., 1995	135 neurologically healthy psychology students	Analyze psychometric properties of the <i>CCST</i>	<p>Found four underlying factors explaining 90% of the variance: accuracy, <i>Shipley's</i>, <i>CCST</i>, and attention.</p> <p>Verbal and non-verbal scores for Free Sorting loaded on independent factors; scores for Structured and Cued Sorting loaded on one factor.</p>	<p>Older age group, no neurological impairment.</p> <p><i>WCST</i> account for 46% of <i>CCST</i> variance (factor 1), with perseverative errors, responses and total errors loading .96 on factor 1.</p>
	Strong et al., 2010	65 pts with complicated	Investigate criterion validity of the	TBI pts scored lower than controls on letter fluency and	Different neurological impairment.

	TBI; 65 age, gender, ethnic, and education matched controls	<i>DKEFS</i> verbal and design fluency subtests	category shifting; design fluency subtests lack criterion validity. Category Switching especially sensitive to TBI functional impairment.	No significant relationship between <i>DKEFS</i> letter fluency or category shifting and perseverative responses on <i>WCST</i> ; suggests <i>DKEFS</i> and <i>WCST</i> measure different aspects of cognitive functioning.
8	Delis, Kaplan, Kramer, 2001 (technical manual)	23 participants, aged 19-78	Comparison of performance on the <i>WCST</i> and <i>DKEFS</i> , correlational analysis	Moderate to high correlations between Number of Categories and various <i>DKEFS</i> subtests (.31-.59); best evidence for use of sorting test

Note. *WCST* = Wisconsin Card Sorting Test; *D-KEFS* = Delis-Kaplan Executive Function System; *MMSE* = Mini Mental State Examination; *FAS* = Fetal Alcohol Syndrome; *FSIQ* = Full Scale IQ; *CCST* = California Card Sorting Test; *TBI* = Traumatic Brain Injury

increased cognitive demand. Reliability coefficients for the *TMT* test-retest reliability range from .70 to .89, with condition B showing greater levels of stability (Strauss et al., 2006). In addition, the results appear to be less stable in clinical groups than in healthy controls. Practice effects can be a concern if the test is given again after a brief interval (three weeks to six months), although longer intervals do not demonstrate the same practice effects. The two forms are correlated to a low extent based on Cohen's criteria, indicating that they assess for similar yet distinct domains. The *TMT* is correlated to a medium extent based on Cohen's criteria with other measures of visual search and scanning abilities, such as the *Symbol Digit Modalities Test* (Royan, Tombaugh, Rees, and Francis, 2004), and correlational studies with independent measures of intelligence (the *WISC-R*, the *Verbal Fluency Test*) suggest that the *TMT* measures something different than intelligence (Strauss et al., 2006).

The *D-KEFS* included the *TMT-A* and *TMT-B* as a part of the Trail Making subtest, under the labels of Number Sequencing and Number-Letter Switching respectively (Delis et al., 2001). In addition, the *D-KEFS* has three other Trails conditions: Visual Scanning, Letter Sequencing, and Motor Speed. Reliability and validity data for the extended Trails condition were not provided by the test creators, nor did a search of the psychological literature reveal any independent studies of *D-KEFS* test characteristics. Delis, Kramer, Kaplan, and Holdnack (2004) defend the lack of reliability and validity information provided in the test manual by pointing out that developing the *D-KEFS* was a long process, during which they published their results in peer-reviewed journals so as to better spread the information. Thus it appears that their argument is that the studies completed on the *TMT* were generalizable to the *D-KEFS* subtests, although this is not clear.

3.2.5. Memory

The sixth area of assessment according to the Canadian guidelines for the diagnosis of FASD is memory. Memory involves a variety of abilities, including the ability to hold information in short term storage to be remembered immediately, to recall over a longer period of time, and to hold and manipulate information for short intervals (Baddeley, 1997). Memory can be verbal, which means involving auditory information, such as words and stories; it can be visual, that is, involving pictures, designs, faces, positions, etc; and it can be physical, involving learned movements and gestures. Some forms of memory are the result of direct teaching and rehearsal, whereas others are implicitly learned without conscious awareness.

For the purpose of these assessments, impairments in memory were defined as deficits as on the *Children's Memory Scale (CMS)*, the *Wechsler Memory Scale, (WMS-III, WMS-IV)*, the *Wide Range Assessment of Memory and Learning, Second Edition (WRAML-2)*, the *California Verbal Learning Task, Second Edition (CVLT-II)*, and the *Rey-Osterrieth Complex Figure Test*. The *CVLT-II* is a task of verbal memory, while the *Rey-Osterrieth* is a measure of visual memory. Each of the other instruments also provides a score for verbal and auditory memory; these scores will be used for comparison purposes. While memory is a complicated function consisting of many elements, only visual and verbal information will be compared for this study, as these are the types of memory routinely assessed in a neuropsychological battery.

The *Children's Memory Scale (CMS)* was developed as an instrument of memory and learning in children aged five to sixteen (Davis, 2010). It consists of nine subtests, six of which are considered core to the assessment of memory, and provides scores on 6 domains: Visual Immediate, Visual Delayed, Verbal Immediate, Verbal Delayed, General Memory, and Attention/Concentration (Cohen, 1997). Tasks require test takers to recite a story from memory, learn an association between a pair of words through repetition, learn a word list through repetition, recall the location of dots over a series of trials, recognize and identify a set of faces from distracters, remember family tableaux, repeat random digit sequences, and recall the location of objects in a picture, among other tasks. For the purposes of our analyses, we will be using the Verbal score on the *CMS* as a representation of verbal memory, and visual memory will be operationally defined as the score on the Visual domain. In both cases, only the immediate conditions will be compared, as other tests, such as the *WRAML*, do not provide a score for delayed memory and so comparisons could not be drawn between individuals assessed on different measures.

The *CMS* was normed on a sample of 1000 children between the ages of five and sixteen, and the sample was selected to most accurately represent the gender, ethnic, and geographic regional make-up of the American population (Cohen, 1997). Estimates of internal reliability range from .70 to .83 in the Visual Immediate condition, and .84 to .90 in the Verbal Immediate condition. Test-retest studies reveal strong stability in Visual Immediate memory in lower age groups, with a corrected correlational coefficient of .69 for children aged five to twelve; for children aged 13 to 16, the correlation was much lower, at .29. In contrast, Verbal Immediate

scores were found to be stable across the age groups, with the corrected coefficient for all groups ranging from .81 to .85.

For all domains, test content of the *CMS* has been revised numerous times based on clinical application, research, and professional review to ensure content validity (Cohen, 1997). The low to moderate correlations between subtests and indexes are evidence that the subtests of the *CMS* have good convergent and divergent validity, and a factor analysis was conducted to ensure the best fit of the underlying model; such issues are related to construct validity. In addition, the subtests of the *CMS* show a moderate to high degree of convergent validity with similar memory measures, such as the *WMS-III* (Visual Immediate for both scales correlated at .55; Verbal Immediate correlated with Auditory Immediate at .74).

The *Wide Range Assessment of Memory and Learning, Second Edition (WRAML-2)* was created as a measure of memory ability, to differentiate between visual, verbal, and global memory impairments (Davis, 2010). It is composed of six core subtests, which provide scores on three domains (Verbal Memory, Visual Memory, and Attention/Concentration), as well as a general memory score (Sheslow & Adams, 2003). There are also optional subtests available to provide a more in-depth memory assessment. It was designed so that clinicians could use it in a fixed or cross battery approach (Davis, 2010), meaning the examiner is able to select some or all of the core subtests, and supplement with optional subtests as one sees fit. Many of the subtests are similar to what is found on the *CMS*. The *WRAML-2* requires that test takers repeat details of an orally administered story, learn a word list over a series of trials, draw geometric images from memory, identify differences between two similar scenes, replicate a visual sequence pattern, repeat a list of objects while ordering those objects in some way (for example, saying a list of animals and non-animals, beginning with all the animals), and repeat back a sequence of letters and numbers. The domain scores for Verbal and Visual Memory will be used for comparison purposed in this study.

The *WRAML-2* was normed on a sample of 1200 individuals, aged five through ninety. Test developers selected the sample with the goal of matching the US census data for 2001 regarding race/ethnicity, gender, geographical region and educational attainment. Tests of internal consistency range from coefficients of .89 to .94 on the Verbal Memory Index, and .82 to .93 on the Visual Memory Index. Using test-retest statistics, both the auditory and visual domains demonstrated good stability, with corrected correlation coefficients at .85 and .67,

respectively. Studies of internal validity indicated that the subtests correlate significantly with one another, but the relationships between subtests are low to moderate. Comparison data on the *WRAML-2* indicate that it correlates significantly with the *WMS-III*, the *CMS*, and the *California Verbal Learning Test*, First and Second Editions (*CVLT-I*; *CVLT-II*). In addition, while the *WRAML-2* was found to correlate with tests of intelligence and achievement, such as the *WAIS-III*, *WISC-III*, and the *WRAT-3*, it was also found to contribute unique information to the assessment of individuals. Moreover, the Verbal Memory and Visual Memory indexes have a strong relationship with corresponding tests of other measures; for example, the tests of visual memory correlated more strongly with the perceptual tasks on the *WAIS* than the verbal tasks.

The *Wechsler Memory Scale*, Third and Fourth Editions (*WMS-III*; *WMS-IV*), are structured slightly differently from one another. The *WMS-III* provides scores on eight domains of memory functioning: Auditory Immediate, Auditory Delayed, Auditory Recognition Delayed, Visual Immediate, Visual Delayed, Working Memory, Immediate Memory, and General Memory (Wechsler, 2009). It consists of ten core subtests similar to those discussed in relation to the *CMS* and the *WRAML-2*; individuals are asked to recall details of a story they were told, learn to pair words together through trials, recite a sequence of numbers, recognize and differentiate a set of faces from distracters, recall the location and activities of family members in a tableau, and replicate a series of physical movements. For the purpose of our analyses, we will be using the Visual Immediate score of the *WMS-III* to operationally define visual memory. We will define verbal memory as scores on the Auditory Immediate domain.

The number of scores provided was lessened from the third to the fourth edition of the *WMS*. The *WMS-IV* supplies only five domain scores: Auditory Memory, Visual Memory, Visual Working Memory, Immediate Memory, and Delayed Memory (Wechsler, 2009). In contrast to the *WMS-III*, the fourth edition contains only six core subtests and one optional subtest. Some sections of the test underwent heavy reworking. For example, the subtests requiring test takers to identify faces were eliminated, as were the set of family tableaus and the physical movement subtests. In their place, subtests involving design and spatial memory were inserted. For the new subtests, test takers were requested to draw a number of designs from memory, recall the location of dots on a grid, and recall a specific set of designs and the location of said designs on a grid. The substance of the verbal tasks was largely unchanged from one edition to the next, although individual items were changed. For the purpose of our analyses, we

will be using the Visual Memory score of the *WMS-IV* to operationally define visual memory. We will define verbal memory by the participant's score on the *WMS-IV* Auditory Memory condition.

The *WMS-III* and *WMS-IV* were normed on similar populations; both samples consisted of a large number of participants, 1250 for the *WMS-III* and 1400 for the *WMS-IV* (Tulsky & Zhu, 1997; Wechsler, 2009). Both groups ranged in age from 16 to 90 years old; as such, the scales can only be administered to individuals over the age of 16. However, only the *WMS-IV* was normed on a sample chosen to best represent the demographic characteristics of the American population as a whole, by attempting to match for gender, race/ethnicity, and level of education (Wechsler, 2009). The *WMS-III* has good internal reliability, with coefficients ranging from .70 to .90 (Tulsky & Zhu, 1997). One concern raised by the test developers is the potential lack of internal validity of the *WMS-III*, as the indexes were not created using factor analysis, but is rather based on “clinically meaningful aspects of clinical memory assessment” (pp. 17). However, it has been shown to correlate significantly with other measures of memory, such as the *Wechsler Memory Scale, Revised* (*WMS-R*) and the *CMS*. Regarding the *CMS*, scores of immediate visual memory correlated at .55 and scores of auditory/verbal memory correlated at .74. This provides evidence for external, concurrent validity.

The *WMS-IV* has shown to have very good internal consistency, with an average correlation coefficient of .95 for the Auditory Memory Index and .96 for the Visual Memory Index (Wechsler, 2009). As well, test-retest coefficients are also high, ranging (by age) from .83 to .87 for the Auditory Memory Index, and .80 to .81 for the Visual Memory Index. Content validity was maintained through comprehensive literature searches and expert review, with modifications to the *WMS-III* stemming from this information in combination with customer feedback and an expert panel. In addition, the *WMS-IV* shows good concurrent validity to other measures of memory. The Auditory Memory Index of the *WMS-IV* correlates significantly with the Auditory Immediate Index, with a correlation coefficient of .81; the Visual Memory Index correlated with the Visual Immediate Index at .50, suggesting comparisons made on verbal memory may be more accurate than those based on visual memory. Likewise, the Verbal Immediate score on the *CMS* correlated with the Auditory Memory Index of the *WMS-IV* at .69, whereas the relationship between the Visual Immediate score on the *CMS* and the Visual

Memory Index of the *WMS-IV* was less strong at .46. The Auditory Memory Index also correlates significantly with the *CVLT-II*, as would be expected given its verbal requirements.

Scores on the *California Verbal Learning Test, Second Edition (CVLT-II)* were examined separately from the other measures of verbal memory. The *CVLT-II* measures recognition and recall of two lists of words over both delayed and immediate memory trials (Delis, Kramer, Kaplan, & Ober, 2000). It was normed on a sample of over 1000 adults, aged 16 to 89, specifically chosen to best represent the actual demographics of the American population ethnically, regionally, and by education level. Estimates of internal consistency are very high for the total sample (.94). In terms of validity, an examination of mean scores for the *CVLT-II*, and its predecessor, the *CVLT*, show a high degree of equivalence between the two measures; correlation coefficients between the two test scores range from .72 to .80. Factor analyses have revealed good internal consistency in the *CVLT-II* as well. The *CVLT* has demonstrated very good construct validity, and as the *CVLT* and the *CVLT-II* show a high degree of concurrent validity, it can be inferred that the *CVLT-II* maintains a similar level of construct validity.

Scores on the *Rey-Osterrieth Complex Figure Test (ROCF)* were analyzed independently of the other visual memory tasks. For this task, individuals are asked to copy a complex visual stimulus while it is in front of them, and then draw the same stimulus from memory after a short and a long delay. This measure was normed on a sample of 505 healthy children and adolescents selected from private and public schools, aged six to seventeen (Meyers & Meyers, 1996). This test has some problems with test-retest reliability because it is a memory test, and the situational demands change after the individual has drawn the figure three times (Cohen and Roman, 2006). However, overall Cohen and Roman conclude that the reliability of the measure was strong, with an interrater reliability of .94. Convergent and discriminant validities were found to range from good to excellent, using normal and brain-injured populations. Construct validity was assessed by comparing scores on the *ROCF* with the *WAIS-R*, as well as with neuropsychological measures that are related and unrelated to memory. Reviewers conclude that it is a sound test of visuoconstructional ability and visuospatial memory.

3.2.6. Language

Expressive language deficits were measured by the *Expressive Vocabulary Test, Second Edition (EVT-2)*, and receptive language impairment was determined by the *Peabody Picture*

Vocabulary Test, Fourth Edition (PPVT-4) or by the Expressive Vocabulary and Oral Expression subtests of the *WIAT-II*.

The *Expressive Vocabulary Test, Second Edition (EVT-2)* allows for a quick evaluation of word retrieval and expressive vocabulary. Test takers are shown a picture and asked to either identify the picture in one word (for example, shown a picture of a carrot and asked “what is this?”) or provide a one word synonym for the picture (for example, a picture of a boulder and asked “what is another word for ‘rock’?”). The *EVT-2* provides a single raw score, which can be converted into a standard score, percentile, normal curve equivalent, or stanine score. The *EVT-2* was normed on an American standardization sample of 3540 examinees, and was stratified to match the most recent U.S. census data with regard to sex, race/ethnicity, geographic region, and educational level (Williams, 2007). The same sample was used in the norming of the *PPVT-4*, allowing for direct comparisons between expressive and receptive vocabulary skills.

Studies of reliability on the *EVT-2* generally reveal a high level of internal consistency, indicating a high degree of item uniformity (Williams, 2007). The split-half reliability for Form A is .94 and for Form B is .93. Coefficient alpha for the *EVT-2* was consistently high at all age groups, averaging .96 on each form, suggesting a consistency between independent sets of items. However, due to the statistical methods employed, test developers warn that the alpha coefficients may be overestimates. Alternate-form reliabilities were consistent across age groups, with a mean reliability of .87. Test-retest correlations range from .94 to .97, indicating performance is stable over time and not greatly influenced by changes in the examinee’s physical or mental state. These results indicate *EVT-2* scores are minimally influenced by measurement error.

Studies of validity indicate the *EVT-2* has good construct validity. Regarding content validity, items were selected on the basis of frequency and commonality, and then subjected to a rigorous review process by content specialists and expert bias reviewers (Williams, 2007). The final pool of items was then determined using statistical properties. As our vocabulary grows with age, this growth should be reflected in the *EVT-2* pattern of change with age. The progression of median GSV scores by age follows the pattern typically seen in measures of crystallized intelligence. Convergent validity is represented by high correlations with the *PPVT-4* ($r = .80-.84$), measures of oral language (generally $r =$ mid .50s to low .80s), the Group Reading

Assessment and Diagnostic Evaluation ($r = .60\text{s}$ and $.70\text{s}$), and the first edition of the *Expressive Vocabulary Test* ($r = .78\text{-.}82$).

The *Peabody Picture Vocabulary Test, Fourth Edition (PPVT-4)* is a test of receptive language abilities. During administration, an individual is shown a page with four pictures on it; the examinee is asked to indicate (either by pointing or saying the number of the picture) which picture best represents the meaning of a word spoken by the examiner. Similarly to the *EVT-2*, the *PPVT-4* provides one raw score that can be converted using tables into a standard score, percentile, normal curve equivalent, or stanine score. The *PPVT-4* was normed on a American standardization sample of 3540 examinees aged 2 years 6 months through 90 years and older, and was stratified to match the most recent U.S. census data with regard to sex, race/ethnicity, geographic region, and educational level as well (Dunn & Dunn, 2007).

Studies of reliability generally reveal a high level of internal consistency for the *PPVT-4*, indicating a high degree of item uniformity. The split-half reliability averages .94 or .95 on each form. Coefficient alpha for the *PPVT-4* was consistently high at all age groups, averaging .97 and .96 for Forms A and B, respectively. This suggests a consistency between independent sets of items. However, due to the statistical methods employed, test developers warn that the alpha coefficients may be overestimates. Alternate-form reliability studies resulted in estimates ranging from .87 to .93, with a mean of .89. Test-retest correlations average a correlation of .93 (range = .92-.96), indicating performance is stable over time and not greatly influenced by changes in the examinee's physical or mental state. These results indicate *PPVT-4* scores are minimally influenced by measurement error.

Studies of validity indicate the *PPVT-4* has good construct validity. Regarding content validity, items were selected that could be illustrated by color drawings and that represented 20 content areas (Dunn & Dunn, 2007). The original item pool, from which this edition drew, was developed through field testing and revision. The current selection process evaluated the previous edition to identify items in need of revision or replacement through an in-house review session with *PPVT-3* users. As our vocabulary grows with age, this growth should be reflected in the *PPVT-4* pattern of change with age. The progression of median GSV scores by age follows the pattern typically seen in measures of crystallized intelligence. Convergent validity is represented by high correlations with the *EVT-2* ($r = .80\text{-.}84$), measures of oral language

(generally $r = .60-.79$), the *Group Reading Assessment and Diagnostic Evaluation* (average $r = .63$), and the third edition of the *PPVT* ($r = .84$).

In earlier assessments, receptive and expressive language skills were operationally defined by scores on the Listening Comprehension and Oral Expression subtests of the *WIAT-II*, respectively. The *WIAT-II* is a comprehensive test battery used to assess academic achievement for ages 4 to 85 (Strauss, Sherman, & Spreen, 2006). Subtests for word reading, reading comprehension, mathematical calculations, mathematical reasoning, listening comprehension, oral expression, and written expression are provided. These achievement measures were normed on an extensive and representative Canadian sample. They are reported to have excellent internal reliability, with subtest reliabilities in the high to very high range (Strauss et al., 2006). The *WIAT-II* also has demonstrated strong correlations with IQ tests, other achievement measures, and moderate correlations with school grades. The test-retest reliability is mostly high to very high, with Written Expression and Oral Expression evidencing adequate reliabilities.

Listening Comprehension requires the processing of incoming verbal information and the ability to demonstrate comprehension through an appropriate response. It is divided into three sections: receptive vocabulary, sentence comprehension, and expressive vocabulary. Examinees are asked to listen to details and select a picture that matched a word or a sentence. They are also required to produce a word that matches an oral description or a picture. Oral Expression asks participants to generate a narrative based on visually presented passages, and provide verbal direction with and without cuing (Smith, 2002). Individuals are required to generate oral language to repeat, describe, categorize, recall, and provide information in order to direct others.

Studies of internal consistency reveal that Listening Comprehension has an average coefficient of .80, with estimates ranging from .71 to .85 across the age groups (Smith, 2002). Internal consistency within Oral Expression is even stronger, with an average coefficient of .86, and estimates ranging from .83 to .89 across the age groups. In regard to test stability, test-retest coefficients ranged from .88 to .93 for Listening Comprehension, with an average coefficient of .91. Oral expression evidenced similar levels of stability, with test-retest estimates ranging from .83 to .88, and an average of .86. In determining validity, test manufacturers compared the scores on the *WIAT-II* to other measures. The Listening Comprehension subtest correlated moderately with the *PPVT-III* (.44). Unfortunately, studies comparing the *EVT-2* with the Oral Expression subtest of the *WIAT-II* could not be located.

Each of these aforementioned tests is in common use in psychological practice throughout North America and is listed as a recommended assessment measure in the appendices to the Canadian guidelines for FASD diagnosis (Chudley et al., 2005).

As previously mentioned, the test protocols varied by the age of the participant, and assessment procedures were updated as new measurements become available. For example, when the *WAIS-IV* was published, it was substituted in the assessment battery for the *WAIS-III*. In order to allow for direct comparisons between participants, regardless of the measures used, analyses were done by domain based on standard deviation scores rather than standard scores by test.

3.2.7. Psychosocial data

In addition, self-reported data were collected at the time of assessment including past and current living circumstances (biological family, adoptive family, foster care, group home, independent living), schooling (regular program, special program, not in school), education history (use of special education resources), and current alcohol and drug use. For some participants, additional reports were provided, such as previous psychological assessments, educational reports, and presentencing reports that included the youth's total criminal history. It did not appear that there was a pattern regarding which participants were sent with additional information; for some, reports accompanied a first-time offender whereas others had extensive criminal histories. Instead it seems that the inclusion of additional information was more contingent on the youth's worker than on youth characteristics. Whenever possible, these reports were used to verify self-reported information. This information was coded and included in the analysis to inform as to how the sociocultural demographics of offenders impact their criminal behaviour and if there are differences in such influence between youths with and without FASD.

3.3. Procedure and Analysis

Participants were drawn from a pre-existing database collected by Dr. Jo Nanson in the course of her private practice. The Youth Court in Saskatoon referred youth to her for neuropsychological assessments between 1998 and 2012. Some of these youth were being screened for possible FASD while others were referred for assessment to help with planning for the youth at risk. Those with a history of prenatal alcohol exposure were seen also by a developmental paediatrician, who examined the youth and, in conjunction with Dr. Nanson's report, provided a diagnosis within the FASD spectrum when appropriate. The referral from the

court included the youth's current charges. At the time of assessment, each participant was asked for permission to use the data collected in the course of assessment for research purposes. They were assured that all information would be used in aggregate, ensuring anonymity. Participants were informed that refusing would hold no negative consequences. Because these are vulnerable youth, extra care was taken to be sure that they understood the consent process. The consent was explained slowly in simple language and they were assured that they may refuse now or in the future to have their data used for research purposes without jeopardizing the rest of the assessment process.

Each individual with a reported history of prenatal alcohol exposure was assessed for FASD using the Canadian Guidelines (Chudley et al., 2005), and were examined by a medical specialist in FASD and given a diagnosis within the IOM criteria (1996) based on the history, physical assessment and the neuropsychological assessment. Approximately half of the sample referred for an assessment (both those with and without a history of PAE) received no diagnosis for FASD (56.3%). The largest diagnostic group represented in this sample was pFAS, which comprised 27.4% of the total sample; ARND was diagnosed in 9.6% of cases and FAS in 6.6% of cases. Of the participants not provided a diagnosis of FASD, 56 were missing reports; they were labelled as undiagnosed not because they were found to lack the criteria, but because no diagnostic information was available in the database. Thus it is not possible to say with certainty that they do not have an FASD. For the purpose of analyses involving comparing FASD to non-FASD offenders, these undiagnosed individuals were removed to minimize the error variance. They were included in other analyses for which the diagnosis was not a variable.

The participants' neuropsychological data were converted to standardized z -scores based on the normative data for each measure and the psychosocial data were coded for the purposes of analysis.

Young offenders with a diagnosis of FASD were compared on the neuropsychological measures of interest to a comparison group of offending youths without FASD. In addition, the sub-diagnoses of FASD were compared to one another to determine if differences in their neurocognitive abilities exist. The neuropsychological data were correlated with the coded sociocultural and crime-related variables in order to examine the relationship between neuropsychological impairment, psychosocial factors and specific criminal activity. Crimes were coded as violent, sexual, property, drug or systemic (breaches) for analysis. Systemic crimes are

those generated by failures within the youth justice system such as failure to attend court, curfew violations, etc. Youth may have multiple offenses in any or all categories.

4. RESULTS

4.1. Missing Data

As the dataset for this project was archival, not all data points for each participant were available. Through the coding of data, three types of missing data became an apparent problem. The first kind of missing data was unsystematic, related to the incompleteness of files; over the years, certain reports or test results were misplaced or removed from the file.

The other two forms of missing data were systematic in nature. The second reason for missing data was related to the participant's ability, or lack thereof, to complete a certain task. For example, there are more scores missing for *TMT-B* than for *TMT-A*, as participants were not administered *TMT-B* if they struggled significantly with *TMT-A*. As well, the *WCST* is well known to be frustrating for examinees (Lezak, 1995); in this project, administration of the *WCST* was discontinued when that frustration became too great in order to maintain rapport for the remainder of the assessment. Of the 149 participants administered the *WCST*, 56 youths (or 38%) discontinued early and so we do not have data for this group. Further, this left only the most capable and patient youths' data included in the database; thus any results involving the *WCST* should be interpreted with extreme caution as they are not likely to be representative of this sample or the population as a whole.

The third form of missing data was due to a change in the assessment battery. As described in greater detail in an earlier section, there has been little professional consensus on the requirements for meeting a diagnosis of FASD, which has led to alterations in the assessment battery over the years. As an example, receptive and expressive language deficits were identified as FASD deficits later than more noticeable impairments such as memory and cognition. As such, the *EVT-2* and the *PPVT-4* were added to the battery after nearly half of the participants had already been assessed. This also accounts for some of the missing psychosocial data, as the specific number of living arrangements by age was not systematically queried in initial assessments.

There is an element of judgment involved in deciding to replace (or not) missing data. It is likely better to not replace values as estimates may skew data or obscure relationships. However, there are cases in which this cannot be avoided, such as with archival data. As recommended by Tabachnick and Fidell (2006), any neurocognitive variable missing more than

five percent of data for this sample was estimated using a regression equation provided by SPSS, Linear Trend at Point.

4.2. Outliers

There were a number of severely impaired youths included in this study, several of whom scored far from the mean. This was a problem, as these outliers created relationships that may not otherwise exist. For example, the sample included very few youths charged with a sexual offense; one of those who had a sexual charge on his order for examination scored 17 standard deviations below the mean on the *TMT-A* test, which was creating a moderate relationship between attentional problems and sexual offending (it was clear this found relationship was due at least partially to the outlier, as this youth was not administered *TMT-B* and no significant relationship between sexual offending and *TMT-B* was found).

In order to control for outliers, all *z*-scores greater than four standard deviations from the mean were recoded as four standard deviations from the mean for the purpose of analysis. It was decided that impairment beyond four standard deviations from the mean would be so severe that differences would not be practically meaningful. This also served to reduce the error variance in analyses.

4.3. Demographic Information

The original proposal had included the entire sample in all analyses. Unfortunately, this was not feasible as 42 of the assessed youths were missing the report with the FASD diagnosis. As it was not possible to say with certainty that these participants either did or did not have a diagnosis, they were excluded from all analyses for which participants with FASD were compared to those without (their data were included in other analyses, for which FASD diagnosis was not considered). The demographics for the entire sample are outlined in Table 2.

The sample for hypothesis 1 consisted of 155 participants, being those for whom a diagnostic report was available. This sample was also used when evaluating any question regarding the difference between participants with and without FASD. Their demographic data have been included in Table 3. The mean age for youth with FASD ($M = 15.26$, $SD = 1.38$) was not statistically significantly different from the mean age of youth without FASD ($M = 15.00$, $SD = 1.48$), $t(153) = 1.14$, $p = .26$. Likewise, there was not a statistically significant difference between groups on gender, $\chi^2(1) = .16$, $p = .69$. There was a significant difference between diagnostic groups regarding ethnicity, $\chi^2(1) = 7.58$, $p = .02$, with the FASD group containing

Table 2: Participant Demographic Information

Demographic Variable		Mean/Count	Percent
Age		15.19	n/a
Gender	Females	45	22.8
	Males	152	77.2
Ethnicity	Aboriginal	156	79.2
	Caucasian	26	13.2
	Other	15	7.6
Special Education	Yes	86	61.4
Resources	No	54	27.4
Foster Care	Yes	132	70.6
	No	55	29.4

significantly higher rates of participants of Aboriginal or First Nations descent than the group without FASD, $t(153) = -2.52, p = .01$.

4.4. Hypothesis 1: Neurocognitive Profile

The first hypothesis stated that youth with a diagnosis of FASD would have a different pattern of deficits on neuropsychological variables than youth without FASD. The data for hypothesis 1 were analysed using profile analysis. Profile analysis is a form of MANOVA that compares groups on a series of variables. Profile analysis provides information on the overall difference between groups. For instance, do young offenders with FASD have lower scores in general than offenders without FASD, regardless of whether or not the profiles are parallel? This is similar to the main effect found in ANOVA. Profile analysis also allows one to test for parallelism; that is, are the group profiles parallel? Parallelism behaves as a test of interaction in profile analysis. In this case, it would indicate whether or not people with FASD have a

Table 3: Participant Demographic Information by Diagnosis

		FASD (n = 84)	No FASD (n = 71)	T/χ^2	Sig.
Mean Age		15.26	15.00	1.14	.26
Gender	Females	20	15	.16	.69
	Males	64	56		
Ethnicity	Aboriginal	71	48	7.58	.02
	Caucasian	7	17		
	Other	6	6		
Special Education	Yes	47	25	2.95	.09
Resources	No	20	21		
Foster Care	Yes	65	43	4.80	.03
	No	16	24		

categorically different pattern of strengths and weaknesses than people without FASD. Finally, if the profiles are found to be parallel, they can be tested for flatness, which examines the similarity in answers to all the dependent variables, regardless of group membership. As a whole, does the sample score high or low on any variables?

One limitation to profile analysis is that all measures must be compared on the same scale. This is not a concern in the present research proposal, as all raw scores were converted to z-scores for comparison purposes. Another limitation is that profile analysis cannot give information on causal relationships. However, it is still possible to speak to the differences that exist (if differences are found to exist).

The measures used to evaluate each domain, as well as the entire sample mean score for each domain, have been included in Table 4.

Table 4: Measures and Means for the Whole Sample

Domain	Measures	N	Mean	St. Dev.
Cognition (FSIQ)	WISC-IV; WAIS-III; WAIS-IV	188	-1.50	.86
Rey Immediate Recall	Rey Complex Figure Test	197	-1.06	1.29
Rey Delayed Recall	Rey Complex Figure Test	197	-1.31	1.16
Verbal Memory 1	CMS; WMS-III; WMS-IV; WRAML-2	197	-1.42	1.06
Visual Memory 1	CMS; WMS-III; WMS-IV; WRAML-2	197	-.70	1.06
Verbal Memory 2	CVLT-2	197	-1.30	1.15
Word Reading	WRAT-3; WRAT-4	197	-1.22	.89
Reading Comprehension	WRAT-3; WRAT-4	197	-1.78	.76
Math Computation	WRAT-3; WRAT-4	197	-1.78	.90
Expressive Vocabulary	WIAT-II; EVT-2	197	-.814	.87
Receptive Vocabulary	WIAT-II; PPVT-4	197	-1.47	.61

Domain	Measures	N	Mean	St. Dev.
Attention 1	TMT-A; D-KEFS (Trails Test, Condition 2)	197	-1.33	1.32
Attention 2	TMT-B; D-KEFS (Trails Test, Condition 4)	197	-2.18	1.49
Verbal Fluency	COWAT; D-KEFS (Letter Fluency)	197	-1.01	.96
Inhibition	WCST	197	.09	.66

It was predicted that youths with FASD would evidence a different pattern of deficits than youths without FASD. Using Hotelling's trace, the profiles were not flat, $V = 8.89$, $F(14, 134) = 85.04$, $p < .001$, indicating that the participants overall performed better on some measures of interest than others. However, these profiles were parallel; there was not a significant main effect of diagnosis on neuropsychological profile using Wilk's Lambda, $V = .92$, $F(14, 134) = .79$, $p = .68$. This suggests that the neuropsychological profile of young offenders with and without an FASD were similar. See Table 5 for a comparison of the means and standard deviations for each domain with the effect size included. Table 6 presents the same data in standard scores format with a mean of ten and a standard deviation of 15, for easier clinical interpretation.

The groups with and without FASD were found to differ in overall severity of cognitive deficits (i.e., the main effect of diagnosis was significant once the neuropsychological variables were collapsed): $F(1, 147) = 19.20$, $p < .001$. Thus it does appear that overall, while their profiles did not significantly differ, the participants without FASD performed better on the neuropsychological measures on the whole than participants with a diagnosis. Figure 1 represents the comparative profiles for the participants with and without FASD.

It was also hypothesized that young offenders with a diagnosis of FAS would be more impaired on neurocognitive measures than participants with a diagnosis of pFAS or ARND, given the perceived severity of the different diagnoses. The profiles for the various FASD

Table 5: Domain Mean Z Scores by Diagnosis

Domain	Diagnosis	Mean (z score)	St. Dev	n	Cohen's <i>d</i>
Cognition (FSIQ)	FASD	-1.63	.77	83	-.32
	No FASD	-1.35	.96	66	
Rey Immediate Recall	FASD	-1.22	1.31	84	-.15
	No FASD	-1.03	1.29	71	
Rey Delayed Recall	FASD	-1.50	1.11	84	-.24
	No FASD	-1.23	1.10	71	
Verbal Memory 1	FASD	-1.67	1.06	84	-.23
	No FASD	-1.43	1.09	71	
Visual Memory 1	FASD	-.93	1.15	84	-.33
	No FASD	-.59	.91	71	
CVLT-2	FASD	-1.49	1.16	84	-.31
	No FASD	-1.06	1.11	71	
Word Reading	FASD	-1.35	.84	84	-.35
	No FASD	-1.04	.91	71	
Reading Comprehension	FASD	-1.88	.63	84	-.50
	No FASD	-1.53	.79	71	
Math Comprehension	FASD	-1.94	.82	84	-.48
	No FASD	-1.52	.96	71	

Domain	Diagnosis	Mean (z score)	St. Dev	n	Cohen's <i>d</i>
Expressive Vocabulary	FASD	-1.05	.74	84	-.73
	No FASD	-.45	.91	71	
Receptive Vocabulary	FASD	-1.57	.56	84	-.54
	No FASD	-1.26	.59	71	
Attention 1	FASD	-1.56	1.27	84	-.29
	No FASD	-1.20	1.29	71	
Attention 2	FASD	-2.48	1.30	84	-.18
	No FASD	-2.10	1.51	71	
Verbal Fluency	FASD	-1.16	.85	84	-.38
	No FASD	-.80	1.05	71	
Inhibition (<i>WCST</i>)	FASD	.09	.69	84	-.11
	No FASD	.17	.71	71	

diagnoses were not found to be flat, as determined by Hotelling's Trace: $V = 8.24$, $F(14, 69) = 40.61$, $p < .001$. Similar to the previous findings for the overall FASD sample, subgroups of participants with FASD do not perform equally on all neuropsychological measures. Using Wilk's Lambda, the profiles were found to be parallel, indicating that those with FAS evidenced a similar pattern of impairment to those with pFAS and ARND: $V = .61$, $F(28, 138) = 1.31$, $p = .11$. Likewise, there were no significant differences between diagnoses once all measures were collapsed: $F(2, 82) = 1.75$, $p = .18$. In this dataset, there was not a statistically significant difference in neuropsychological profile severity or pattern for subdiagnoses of FASD. Figure 2 displays the various FASD profiles for the neurocognitive data. See Table 7 for a comparison of the means and standard deviations for each domain with the effect size included. The finding that there is no statistically significant difference in profile or severity between sub-diagnoses of

Table 6: Domain Mean Standard Scores by Diagnosis

Domain	FASD (n = 84)	Non FASD (n = 71)	Difference (in SS)
Cognition (FSIQ)	76	80	4
Rey Immediate Recall	82	85	3
Rey Delayed Recall	78	82	4
Verbal Memory 1	75	79	4
Visual Memory 1	86	91	5
Verbal Memory 2 (<i>CLVT-2</i>)	78	84	6
Word Reading	80	84	4
Reading Comprehension	72	77	5
Math Comprehension	71	77	6
Expressive Vocabulary	84	93	9
Receptive Vocabulary	76	81	5
Attention 1	79	85	6
Attention 2	67	72	5
Verbal Fluency	83	88	5
Inhibition (<i>WCST</i>)	101	103	2

Note. SS = Standard score; standard scores have a mean of 100 and a standard deviation of 15

FASD does not mean that a difference between groups does not exist. It is possible that this is a problem of power, as this analysis contained fewer participants in one group than there are variables of interest. This sample consisted of only 13 youths diagnosed with FAS and the profile contains 15 variables. Thus, there may be significant differences between groups that were not captured in this study due to issues of power. In addition, while a p value of .11 does not reach a level of statistical significance as predetermined for this study, it is not far off either, which suggests the possibility that a difference might be found with another sample, with a larger sample, or under different methodological conditions.

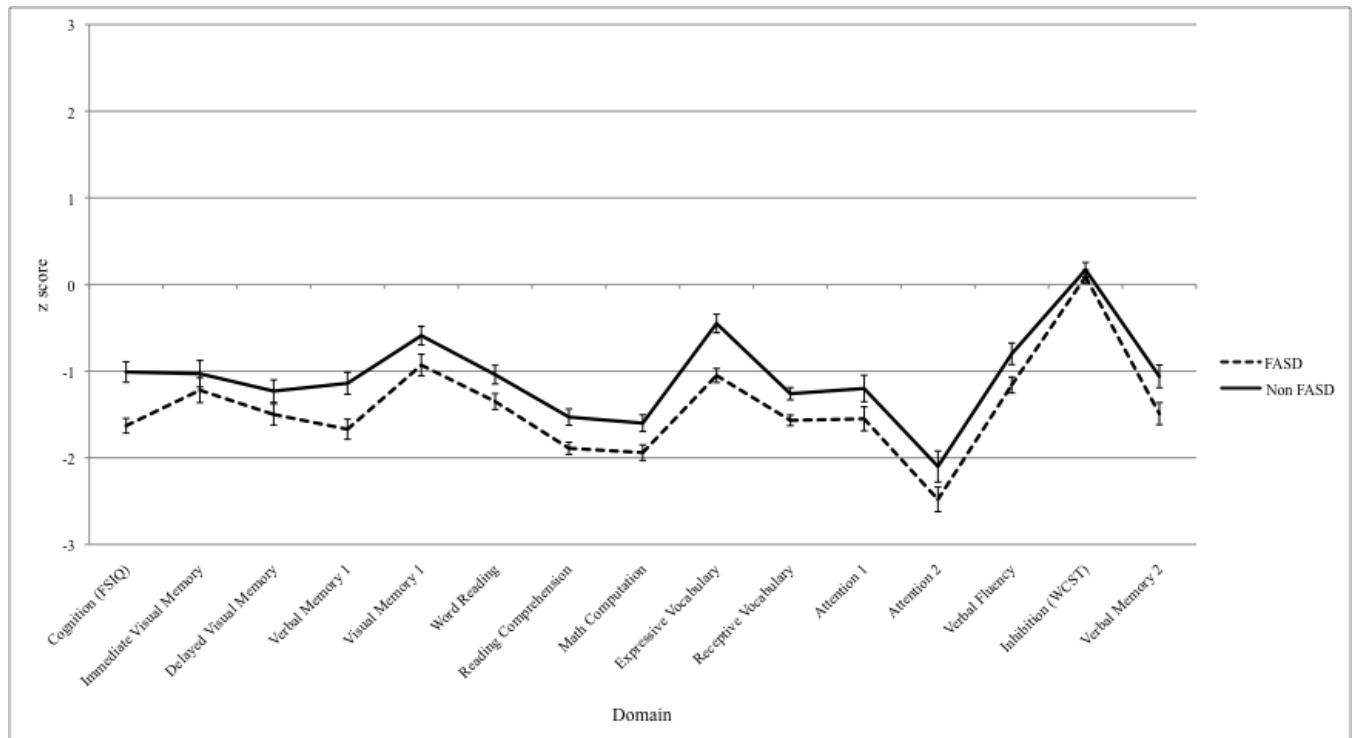


Figure 1. Mean score on each neurocognitive domain by diagnostic group. Youth with FASD did not differ from youth without FASD on profile but they were more severely impaired on the neurocognitive measures across the board. Means are expressed in z score format; vertical lines depict standard errors of the mean.

4.4.1. Post Hoc Analyses

As only 62% of the participants who were administered the *WCST* were able to complete the task, it was hypothesized that these participants might be somehow different from those who discontinued the test due to frustration. Specifically, it was thought that those capable of finishing the *WCST* may be more cognitively capable overall and that this might impact the differences identified between young offenders with and without FASD. To explore this possibility, profile analysis was run again, this time on two groups: those who completed the *WCST* and those who discontinued (participants who were not administered the *WCST* were omitted from these analyses).

For the participants who completed the *WCST*, there was not found to be a significant difference in profile between those with and without FASD using Wilks' Lambda, $V = .81$, $F(14, 56) = .92$, $p = .55$. However, there was a main effect of diagnosis once the measures were collapsed, $F(1, 69) = 10.49$, $p = .002$, suggesting that of the young offenders who complete

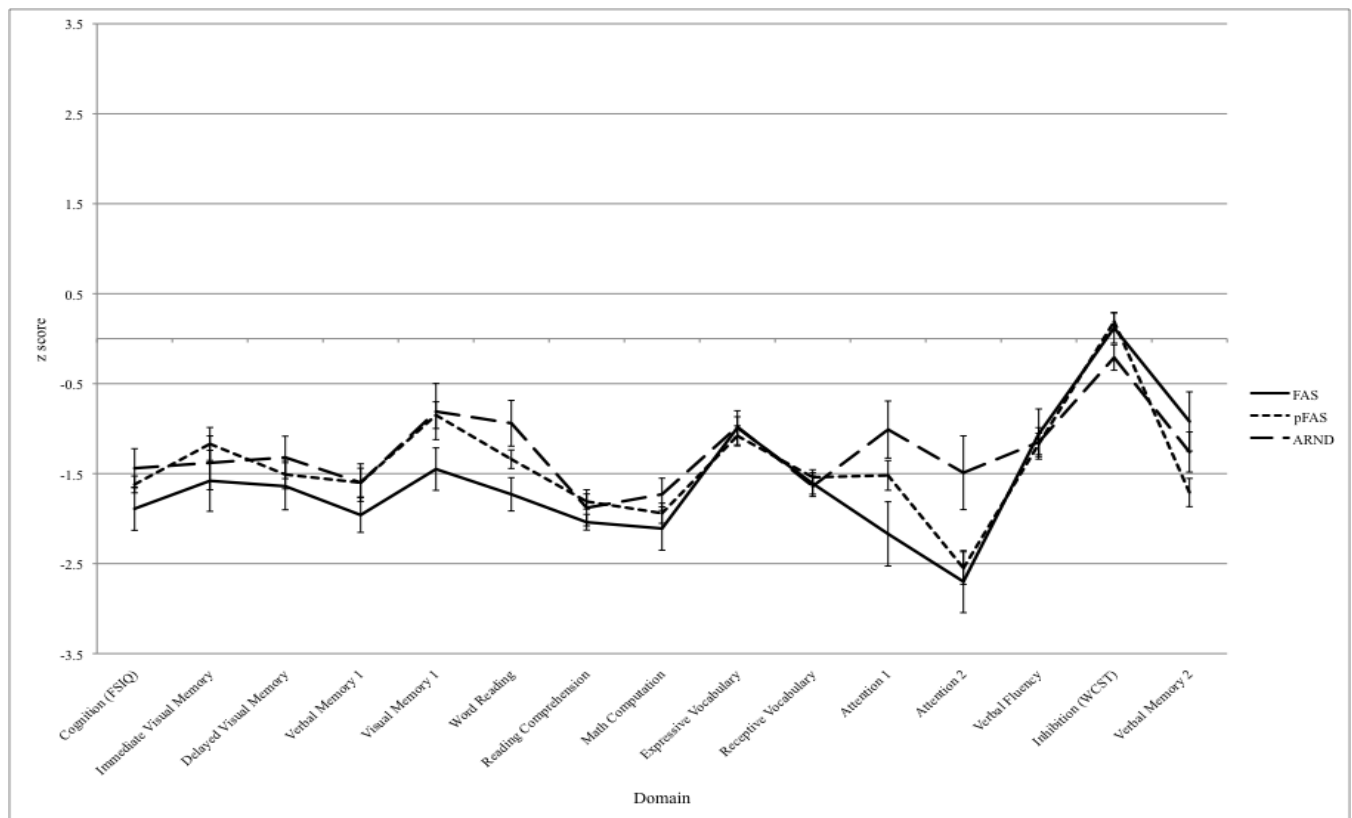


Figure 2. Mean score on each neurocognitive domain by subdiagnostic group. There was no difference found in profile or severity of neurocognitive impairments, although the sample lacked power and differences may have been obscured by this fact. Means are expressed in *z* score format; vertical lines depict standard errors of the mean.

the *WCST*, participants with FASD were more impaired on the neurocognitive measures than participants without FASD. The mean and standard deviation for each of the variables is presented in Table 8 and the profiles are represented in graphic form in Figure 3.

Of the youth who discontinued the *WCST* due to inability to grasp the concept or tolerate the frustration well, there was similarly no difference found between the neurocognitive profile of youth with FASD and those without using Wilks' Lambda, $V = .75$, $F(13, 31) = .80$, $p = .65$. However, in this case, there was also no significant difference between diagnostic groups once the measures had been collapsed, although the difference is nearing statistical significance, $F(1, 43) = 3.33$, $p = .08$. The mean and standard deviation for each of the variables is presented in Table 9 and the profiles are represented in graphic form in Figure 4.

Table 7: Domain Mean Z Scores by Subdiagnosis

Domain	Diagnosis	Mean (z score)	St. Dev	n	Eta Squared
Cognition (FSIQ)	FAS	-1.89	.83	12	.03
	pFAS	-1.62	.68	54	
	ARND	-1.44	.95	19	
Rey Immediate Recall	FAS	-1.59	1.22	13	.01
	pFAS	-1.17	1.34	54	
	ARND	-1.38	1.30	19	
Rey Delayed Recall	FAS	-1.64	.95	13	.01
	pFAS	-1.51	1.17	54	
	ARND	-1.33	1.03	19	
Verbal Memory 1	FAS	-1.96	.70	13	.02
	pFAS	-1.60	1.18	54	
	ARND	-1.61	.91	19	
Visual Memory 1	FAS	-1.45	.85	13	.04
	pFAS	-.85	1.09	54	
	ARND	-.81	1.36	19	
CVLT-2	FAS	-.92	1.19	13	.07
	pFAS	-1.71	1.16	54	
	ARND	-1.26	.97	19	
Word Reading	FAS	-1.73	.67	13	.08
	pFAS	-1.35	.76	54	
	ARND	-.94	1.11	19	

Domain	Diagnosis	Mean (z score)	St. Dev	n	Eta Squared
Reading Comprehension	FAS	-2.04	.31	13	.02
	pFAS	-1.81	.62	54	
	ARND	-1.88	.88	19	
Math Comprehension	FAS	-2.11	.87	13	.02
	pFAS	-1.94	.83	54	
	ARND	-1.73	.78	19	
Expressive Vocabulary	FAS	-1.00	.68	13	.004
	pFAS	-1.08	.82	54	
	ARND	-.98	.49	19	
Receptive Vocabulary	FAS	-1.61	.43	13	.01
	pFAS	-1.54	.61	54	
	ARND	-1.64	.50	19	
Attention 1	FAS	-2.17	1.29	13	.07
	pFAS	-1.52	1.22	54	
	ARND	-1.01	1.38	19	
Attention 2	FAS	-2.70	1.24	13	.10
	pFAS	-2.55	1.32	54	
	ARND	-1.49	1.79	19	
Verbal Fluency	FAS	-1.06	1.01	13	.002
	pFAS	-1.17	.87	54	
	ARND	-1.15	.70	19	
Inhibition (WCST)	FAS	.12	.61	13	.06
	pFAS	.19	.70	54	
	ARND	-.21	.62	19	

Table 8. Domain Mean Z Score, Participants who Completed the WCST

Domain	Diagnosis	Mean (z score)	St. Dev	n	Cohen's <i>d</i>
Cognition (FSIQ)	FASD	-1.43	.81	38	-.62
	No FASD	-.95	.72	33	
Rey Immediate Recall	FASD	-.99	1.49	38	-.18
	No FASD	-.72	1.46	35	
Rey Delayed Recall	FASD	-1.33	1.33	38	-.27
	No FASD	-1.00	1.16	35	
Verbal Memory 1	FASD	-1.56	1.22	38	-.57
	No FASD	-.89	1.14	35	
Visual Memory 1	FASD	-.67	.99	38	-.19
	No FASD	-.47	1.05	35	
CVLT-2	FASD	-1.40	1.17	38	-.59
	No FASD	-.74	1.07	35	
Word Reading	FASD	-1.17	.84	38	-.34
	No FASD	-.86	.92	35	
Reading Comprehension	FASD	-1.79	.58	38	-.43
	No FASD	-1.49	.80	35	
Math Comprehension	FASD	-1.80	.79	38	-.40
	No FASD	-1.48	.79	35	

Domain	Diagnosis	Mean (z score)	St. Dev	n	Cohen's <i>d</i>
Expressive Vocabulary	FASD	-1.07	.73	38	-.96
	No FASD	-.20	1.06	35	
Receptive Vocabulary	FASD	-1.49	.59	38	-.56
	No FASD	-1.14	.65	35	
Attention 1	FASD	-1.16	.98	38	-.13
	No FASD	-1.01	1.35	35	
Attention 2	FASD	-2.30	1.23	38	-.22
	No FASD	-1.98	1.67	35	
Verbal Fluency	FASD	-.97	.68	38	-.45
	No FASD	-.63	.84	35	
Inhibition (WCST)	FASD	.12	.98	38	-.15
	No FASD	.27	.96	35	

4.5. Hypothesis 2: Neurocognitive Scores and Offending

The second hypothesis stated that neurocognitive impairment would be related to the types of charges accrued. Specifically, it was predicted that lower scores on measures of memory would be related to a high number of charges related to systemic offenses such as breached conditions and failures to appear. In addition, it was expected that impairment in executive functioning domains (attention, inhibition, verbal fluency) would be related to violent and sexual offenses. All analyses run to explore the second hypothesis were correlational in nature.

4.5.1. Hypothesis 2a

There was no significant relationship between the systemic charges accrued and impairment in memory functioning on any of the memory instruments. Verbal Memory 1, operationally defined as the verbal score on the administered edition of the *CMS*, *WRAML* or *WMS*, was not significantly correlated with index systemic charges, $r(n = 191) = -.09, p$ (one

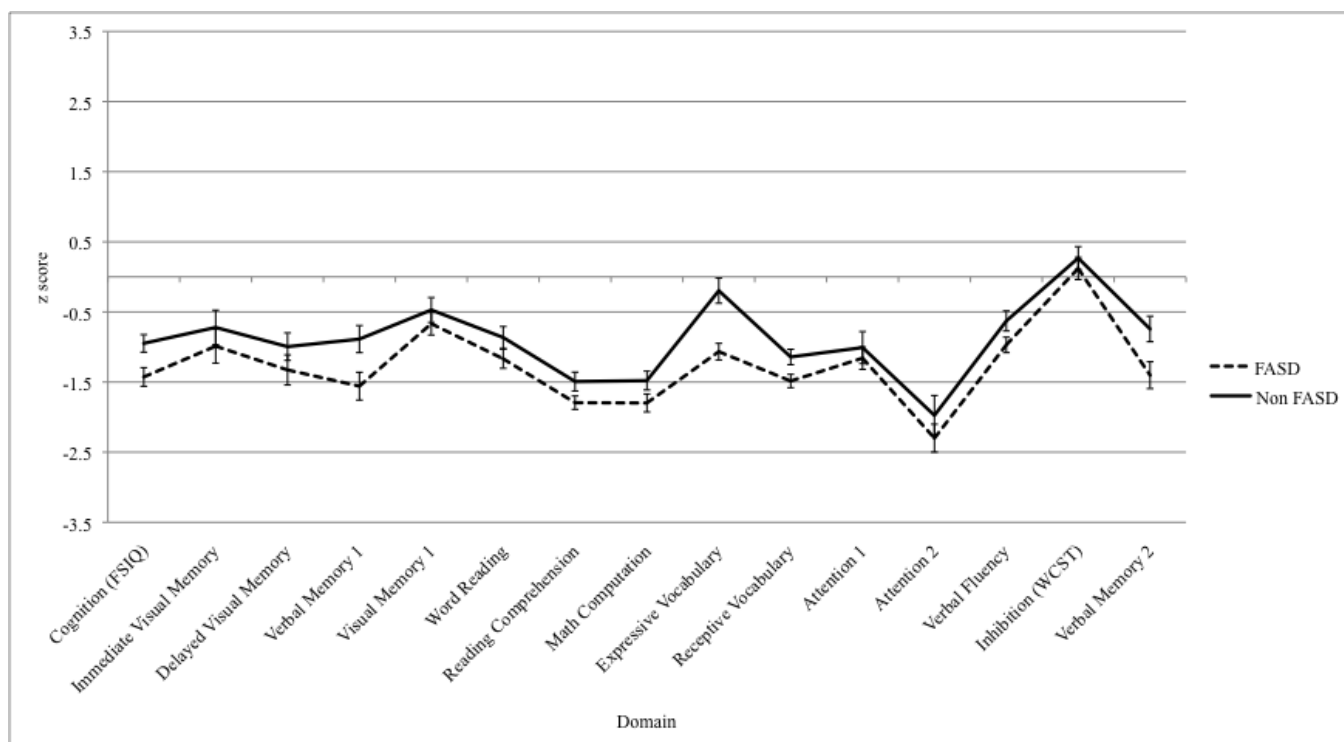


Figure 3. Mean score on each neurocognitive domain by diagnostic group for participants who completed the *WCST*. Youth who completed the *WCST* with FASD did not differ from youth without FASD on profile but they were more severely impaired on the neurocognitive measures across the board. Means are expressed in *z* score format; vertical lines depict standard errors of the mean.

tailed) = .11, nor was it significantly correlated with total system charges, $r(N = 197) = -.13$, p (one tailed) = .04. [Note: while this would be typically be considered significant, the relationship does not hold once a Bonferonni correction is applied to control for Type I errors. A Bonferonni correction in this instance sets the required probability level for significance at .005. A Bonferonni correction is a very conservative method of controlling for Type I errors, and thus may lead to the acceptance of the null hypothesis when it is erroneous. However, given the nature of the data and the inability to account for many variables in real world research, it was decided that it was important to be conservative in the interpretation of findings. In addition, r is an indication of effect size in and of itself and can be judged on its merits without relying on the p value to determine significance (Nickerson, 2000).] A second measure of verbal memory, represented by scores on the *CVLT-2*, was similarly not significantly related to current charges, r

Table 9. Domain Mean Z Score, Participants who Discontinued WCST

Domain	Diagnosis	Mean (z score)	St. Dev	n	Cohen's <i>d</i>
Cognition (FSIQ)	FASD	-1.72	.52	31	.25
	No FASD	-1.89	.84	14	
Rey Immediate Recall	FASD	-1.47	1.30	32	.19
	No FASD	-1.70	1.03	16	
Rey Delayed Recall	FASD	-1.66	.97	32	.04
	No FASD	-1.70	1.05	16	
Verbal Memory 1	FASD	-1.89	.93	32	-.30
	No FASD	-1.58	1.11	16	
Visual Memory 1	FASD	-1.10	1.20	32	-.48
	No FASD	-.58	.94	16	
CVLT-2	FASD	-1.58	1.31	32	-.27
	No FASD	-1.24	1.26	16	
Word Reading	FASD	-1.46	.65	32	-.17
	No FASD	-1.35	.68	16	
Reading Comprehension	FASD	-2.00	.55	32	-.30
	No FASD	-1.85	.45	16	
Math Comprehension	FASD	-2.02	.84	32	-.12
	No FASD	-1.92	.81	16	
Expressive Vocabulary	FASD	-.96	.80	32	-.50

Domain	Diagnosis	Mean (z score)	St. Dev	n	Cohen's <i>d</i>
Receptive Vocabulary	No FASD	-.62	.58	16	-.32
	FASD	-1.60	.55	32	
	No FASD	-1.41	.62	16	
Attention 1	FASD	-2.04	1.28	32	-.43
	No FASD	-1.49	1.29	16	
Attention 2	FASD	-2.85	1.37	32	-.11
	No FASD	-2.71	1.26	16	
Verbal Fluency	FASD	-1.23	.92	32	-.25
	No FASD	-.91	1.62	16	

($n = 191$) = $-.07$, p (one tailed) = $.19$, or total charges, r ($N = 197$) = $-.04$, p (one tailed) = $.30$.

The same results stand for the relationships between the measures of visual memory and systemic charges. Visual Memory 1, defined as a participant's visual score on the administered edition of the *CMS*, *WRAML* or *WMS*, was unrelated to current system charges, r ($n = 191$) = $-.05$, p (one tailed) = $.25$ and total system charges, r ($N = 197$) = $.02$, p (one tailed) = $.38$. Scores of Immediate Visual Memory, as defined by the immediate trial score on the *Rey-Osterrieth Complex Figure*, were not related to current system charges, r ($n = 191$) = $-.06$, p (one tailed) = $.23$ nor were they significantly correlated with total system charges, r ($N = 197$) = $.02$, p (one tailed) = $.40$. Delayed Visual Memory, represented by the delayed trail on the *ROCF*, was similarly unrelated to both current system charges, r ($n = 191$) = $-.06$, p (one tailed) = $.20$ and total system offenses, r ($N = 197$) = $.05$, p (one tailed) = $.24$.

All the correlation coefficients between memory scores and number of systemic charges generated have been included in Table 10.

4.5.2. Hypothesis 2b

There was no significant relationship between sexual crimes and the various areas of executive functioning found in this sample. Participants were rated as either having a sexual

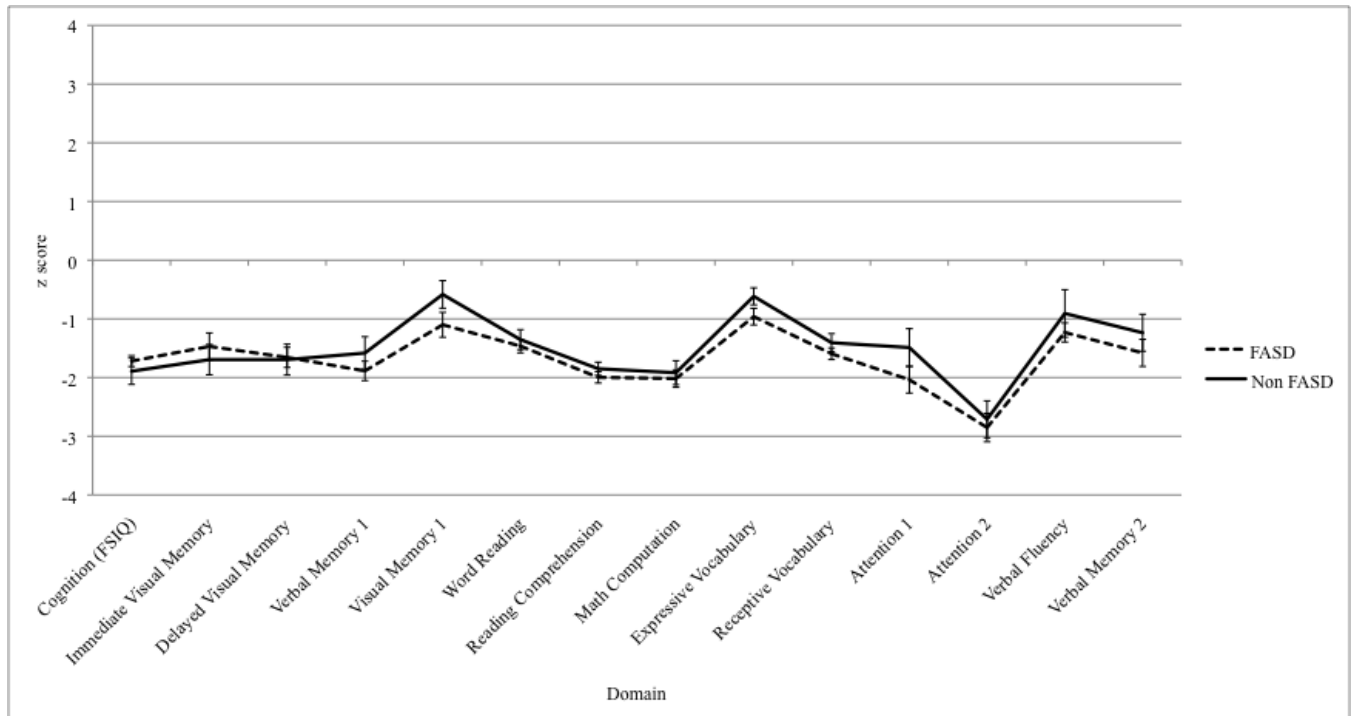


Figure 4. Mean score on each neurocognitive domain by diagnostic group for participants who discontinued the *WCST*. Youth who discontinued the *WCST* with FASD did not differ from youth without FASD on profile or severity on the neurocognitive measures across the board. Means are expressed in *z* score format; vertical lines depict standard errors of the mean.

charge or not and point biserial correlations were run to determine if a relationship was present. There was no correlation between inhibition, as defined by the number of perseverative errors made on the *WCST*, and the number of index sexual charges accrued, $r_{pb} (N = 197) = .08$, p (two tailed) = .25, or total sexual charges, $r_{pb} (N = 197) = .06$, p (two tailed) = .38. Similarly, verbalfluency was unrelated to both current sexual charges, $r_{pb} (N=197) = .01$, p (two tailed) = .87, and total sexual charges, $r_{pb} (N = 197) = .03$, p (two tailed) = .66.

The relationship between attention and sexual offending in this sample was slightly more complex. There was a minor significant correlation found between current sexual charges and a simple measure of attention (Attention 1), as defined by Trails A or the second condition of the *D-KEFS* Trails Test, $r_{pb} (N = 197) = .19$, p (two tailed) = .009. However, with the number of comparisons performed in this analysis, a Bonferonni correction sets the required probability

Table 10. Correlation Between Memory Scores and Systemic Charges

	Verbal Memory 1	Verbal Memory 2	Visual Memory 1	Visual Short- term Memory	Visual Long- term Memory
Current System Charges	-.09	-.07	-.05	-.06	-.06
Total System Charges	-.13	-.04	.02	.02	.05

level for significance at .006, rendering this correlation statistically insignificant. As mentioned before, the r value can stand on its own merits; in this case, the relationship appears to be slight, accounting for 4% of the variance in the relationship, $r^2 = .04$. There was no significant relationship between current sexual charges and a more complex measure of attention (Attention 2), defined as Trails B or the fourth condition of the *D-KEFS* Trails Test, $r_{pb} (N = 197) = .12$, p (two tailed) = .08. Likewise, there were no significant relationships between total sexual offending charges and Attention 1, $r_{pb} (N = 197) = .14$, p (two tailed) = .05, or Attention 2, $r_{pb} (N = 197) = .09$, p (two tailed) = .20.

All the correlation coefficients between executive functioning scores and number of sexual charges generated have been included in Table 11.

4.5.2.1. Post-hoc Analyses: 2b. As the number of young offenders with a sexual charge was very small in this sample, there was concern that a few participants with extreme scores might be creating a relationship with attention that may or may not be present in the real world. As such, a receiver operating characteristic (ROC) curve was run to determine if classification as a sex offender could be reliably determined using the score on Attention 1 (the only measure with which sex offenders demonstrated a meaningful relationship). As determined by the area under the curve (AUC; $N = 197$) = .68, 95% CI [.50, .85], classification of sex offenders based on this one measure resulted in a moderate to large effect size. However, it is important to keep in mind the severely limiting size of the sample in drawing conclusions based on this analysis. The ROC curve graph is presented in Figure 5.

Table 11. Correlation Between Executive Functioning Scores and Sexual Charges

	Attention 1	Attention 2	Inhibition (WCST)
Current Sexual Charges	.19	.12	.08
Total Sexual Charges	.14	.09	.06

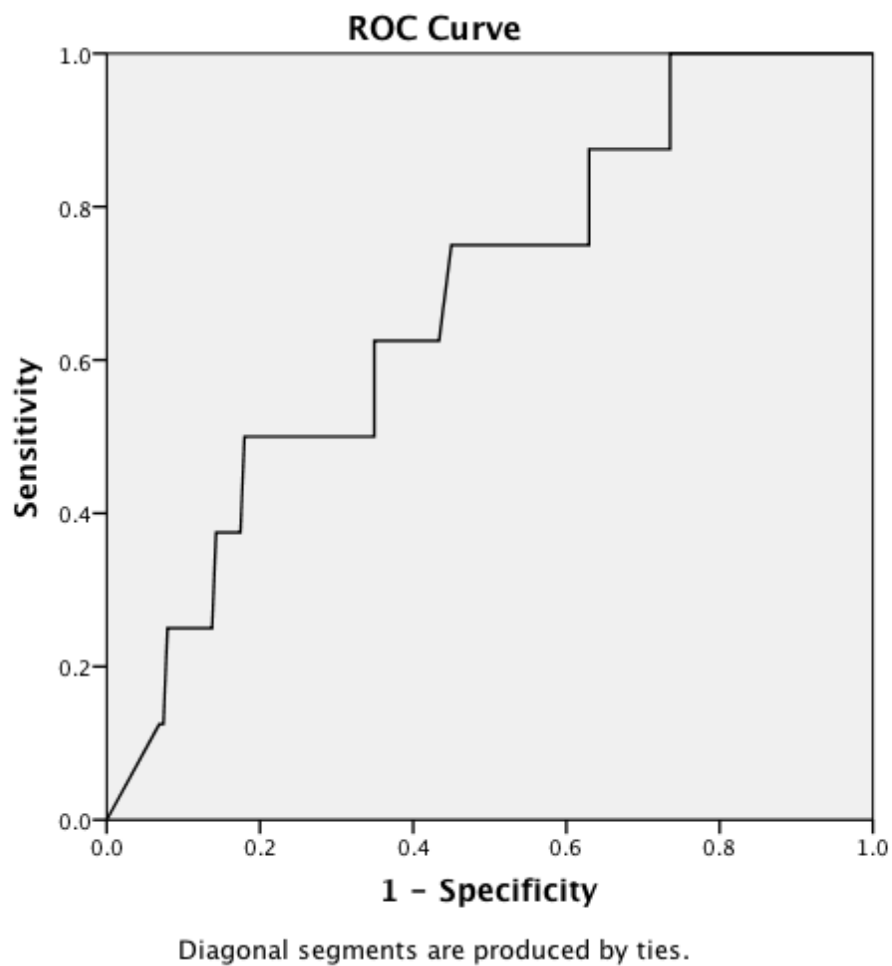


Figure 5. Receiver operating characteristic (ROC) curve classification of sex offenders by score on Attention 1. The area under the curve (AUC) indicates that using Attention 1 scores to identify offenders with a sexual charge results in a moderate to large effect. This finding must be interpreted with caution as the sample size for offenders with a sexual charge is very small.

4.5.3. Hypothesis 2c

Participants were rated as either having a violent charge or not and a point biserial correlation was run to determine if a relationship was present. Akin to the results found for sexual offending, there did not appear to be any significant relationship between violent offense charges and the measures of executive functioning in this sample. Current violent charges were unrelated to inhibition, as defined by the number of perseverative errors made on the *WCST*, r_{pb} ($N = 197$) = .04, p (two tailed) = .59. Total violence charges were similarly unrelated to inhibition, r_{pb} ($N = 197$) = -.15, p (two tailed) = .03. This relationship was not considered significant because a Bonferonni correction applied to this data set the required probability level for significance at .006 and the correlation coefficient indicates that the relationship is small at best.

It is possible that the Bonferonni correction has resulted in a Type II error here, as a correlation coefficient of -.15 suggests a possible small relationship. If that is the case, then it could be expected that as the number of perseverative errors made increases, the number of charges accrued decreases. This is the opposite of what one would predict, given that fewer perseverative errors are indicative of better capacity to inhibit; however, ultimately this finding is biased as only the most cognitively capable youths were able to complete the task and thus garner a score to be reported.

There was no significant relationship found between verbal fluency and participants with current violent charges, r_{pb} ($N = 197$) = -.06, p (two tailed) = .41, or total violent charges, r_{pb} ($N = 197$) = .01, p (two tailed) = .90.

Likewise, this sample did not demonstrate any relationship between violent offending and attention. There was no relationship between current charges of violence and Attention 1, r_{pb} ($N = 197$) = .05, p (two tailed) = .46, or Attention 2, r_{pb} ($N = 197$) = -.02, p (two tailed) = .83. When total violent charges were taken into account, the relationship between violence and Attention 1 remained insignificant, r_{pb} ($N = 197$) = -.03, p (two tailed) = .64, as did the relationship between total violent charges and Attention 2, r_{pb} ($N = 197$) = .004, p (two tailed) = .96.

All the correlation coefficients between executive functioning scores and number of violent charges generated have been included in Table 12.

4.5.4. Hypothesis 2d

It was hypothesized that young offenders with FASD would evidence stronger

Table 12. Correlation Between Executive Functioning Scores and Violent Charges

	Attention 1	Attention 2	Inhibition (WCST)
Current Violent Charges	.05	-.02	.04
Total Violent Charges	-.03	.01	-.15

relationships between neuropsychological impairment and types of crime committed than young offenders without FASD as the deficits in FASD were hypothesized to be linked to such individual's criminal involvement. Data for hypothesis 2d were analyzed by converting the difference in correlational coefficients into a Fisher's Z score and comparing this score to a normal distribution to determine if the difference is significant. Fisher's Z is a statistic useful for assessing the significance of the difference between two correlation coefficients found in independent samples (Field, 2009). Table 13 outlines the various correlational relationships between the types of charges accrued by and the demonstrated neurocognitive deficits of the participants, as differentiated by the presence of a diagnosis of FASD.

As evidenced in Table 13, there were not found to be statistically significant group differences between the correlation coefficients on any of the proposed relationships between neurocognitive deficits and types of crime committed. In this sample, the deficits resulting from FASD do not appear to be more related to criminal behaviour than the deficits displayed by the group without FASD.

4.5.5. *Post Hoc Analyses*

As the expected relationships between specific neurocognitive impairments and types of crime were not found to be significant, it was hypothesized that neurocognitive deficits on the whole may be related to an increased risk of criminal activity. As such, a number of further correlational analyses were run to determine if any measures employed were related to the general number of charges accrued, both for current and total charges. The results have been included in Table 14. A Bonferroni correction placed the level of significance at .003.

In regard to current charges, there were two significant associations between neurocognitive deficits and charges generated, one of which remains significant following the

Table 13: Difference in Correlation Between Type of Crime and Neurological
Impairment by Diagnostic Group

Hypothesized Relationship	FASD r (N = 84)	Non FASD r (N = 71)	Fisher's Z	Sig. (two- tailed)
Verbal Memory 1 and Current System Charges	-.01	-.12	.67	.50
Verbal Memory 1 and Total System Charges	-.13	-.12	-.06	.95
Verbal Memory 2 and Current System Charges	.16	-.14	1.84	.07
Verbal Memory 2 and Total System Charges	.13	-.17	1.84	.07
Visual Memory 1 and Current System Charges	.03	-.06	.55	.58
Visual Memory 1 and Total System Charges	.05	.01	.24	.81
Immediate Visual Memory and Current System Charges	.01	-.12	.79	.43
Immediate Visual Memory and Total System Charges	.03	.03	.00	1.00
Delayed Visual Memory and Current System Charges	-.03	-.05	.12	.90
Delayed Visual Memory and Total System Charges	.02	.08	-.37	.71

Hypothesized Relationship	FASD r (N = 84)	Non FASD r (N = 71)	Fisher's Z	Sig. (two- tailed)
Attention 1 and Current Sexual Offense Charges	-.18	-.12	-.37	.71
Attention 1 and Total Sexual Offense Charges	-.18	.07	-1.53	.13
Attention 2 and Current Sexual Offense Charges	-.09	-.13	.25	.80
Attention 2 and Total Sexual Offense Charges	-.09	-.02	-.43	.67
Inhibition and Current Sexual Offense Charges	.04	.05	-.06	.95
Inhibition and Total Sexual Offense Charges	.04	.01	.18	.86
Attention 1 and Current Violent Offense Charges	-.13	-.10	-.18	.86
Attention 1 and Total Violent Offense Charges	-.04	-.04	.00	1.00
Attention 2 and Current Violent Offense Charges	-.05	.10	-.91	.36
Attention 2 and Total Violent Offense Charges	-.08	-.06	-.12	.91
Inhibition and Current Violent Offense Charges	-.01	.14	-.92	.36
Inhibition and Total Violent Offense Charges	-.18	-.15	-.19	.85

Table 14. Correlation Between Neurocognitive Scores and Charges Accumulated

Domain (N = 197)	Current Crimes (n = 191)	Total Crimes (N = 197)
Cognition (FSIQ)	-.06	.04
Rey Immediate Recall	.02	.06
Rey Delayed Recall	-.02	.08
Verbal Memory 1	-.08	-.13
Visual Memory 1	-.03	.00
Verbal Memory 2 (CLVT-2)	-.10	-.05
Word Reading	.09	.05
Reading Comprehension	-.02	-.03
Math Comprehension	-.20**	-.08
Expressive Vocabulary	.07	-.07
Receptive Vocabulary	.03	-.04
Attention 1	-.08	.05
Attention 2	-.15*	-.02
Verbal Fluency	-.05	-.11
Inhibition (WCST)	.03	.06

* $p < .05$ (2 tailed)** $p < .01$ (2 tailed)

application of the Bonferonni correction. Math comprehension was found to significantly correlate with current charges, $r(n = 191) = -.20$, p (two-tailed) = .007, so that higher math comprehension scores were associated with fewer charges. However, current charges could only account for 4% of the variance in math comprehension scores, $r^2 = .04$. Attention 2 scores, while correlated to a low extent with current charges, $r(n = 191) = -.15$, p (two-tailed) = .04, did not remain significant after a Bonferonni correction. Akin to the results of the association with math computation, better performance on the attention task is related to accumulating fewer charges.

When total number of charges accrued were taken into account, there were no significant correlations between neurocognitive impairment and charges generated.

4.6. Hypothesis 3: Home Stability and Offending

The third hypothesis anticipated that there would be a connection between criminal activity and the participant's environmental stability, as defined by number of living arrangements for three age ranges: ages 0 to 7, ages 8 to 12, and ages 13 to 18 (or age at the time of assessment). These age ranges were chosen based on the work of Striessguth et al. (1997), which specified the ages between 8 and 12 years old to be a particularly vulnerable period for youth who become involved in the justice system.

4.6.1. Hypothesis 3a

It was predicted that long-term stability, as evidenced by living in one location, would be related to fewer crimes.

4.6.1.1. *Hypothesis 3.a.1.* The data for hypothesis 3.a.1 were analyzed using a one-way ANOVA using the coding categories of 1 placement, 2 to 5 placements, and more than 5 placements. There was no significant relationship linking environmental disruption between the ages of 0 and 7 and current charges accrued, $F(2, 151) = .31, p = .74$, partial $\eta^2 = .004$. However, once total number of charges accrued were taken into account, there was a significant, if small, effect of environmental stability from ages 0 to 7 on criminal activity, $F(2, 154) = 4.12, p = .02$, partial $\eta^2 = .05$. There was a significant difference in total criminal activity between participants who lived in one placement ($M = 13.04, SD = 7.48$) and those who lived in two to five homes ($M = 17.91, SD = 12.43$), $p = .01$; those who were placed in more than one home generated significantly more charges. There was no significant difference between youths who lived in one home and those who lived in more than five ($M = 15.94, SD = 8.58$), $p = 1.00$; nor was there a difference between those living in two to five and those living in more than five placements, $p = 1.00$.

When the data were coded, they were coded on the conservative side; that is, youth were only coded as having been placed in greater than five homes if it was explicitly stated (for example, if a report noted that a youth was placed in multiple homes, this was coded as less than five placements unless five were specifically noted). Because of this conservatism, it was decided that it would also be useful to contract the categories and examine the difference between having lived in only one placement and having lived in more than one. A t-test was performed on the data, with results similar to the results of the ANOVA. For the age range of 0 to 7 years old, there was no significant difference in current charges between those who had

lived consistently in one home ($M = 5.82$, $SD = 5.55$) and those who were placed in more than one home ($M = 7.32$, $SD = 6.18$), $t(148) = -.93$, $p = .36$. There was a significant difference between those with one placement ($M = 14.06$, $SD = 11.16$) and those with more than one ($M = 16.16$, $SD = 7.98$) when total number of charges were considered, $t(153) = -2.74$, $p = .007$. Again, those with more placements were more like to have accrued more charges.

4.6.1.2. Hypothesis 3.a.2. The data for hypothesis 3.a.2 were similarly analyzed using a one-way ANOVA using the coding categories of 1 placement, 2 to 5 placements, and more than 5 placements. There was not a significant main effect of home stability on current charges for the age range of 8 to 12 years old, $F(2, 147) = 1.42$, $p = .25$, partial $\eta^2 = .02$. Once total charges were taken into account, there remained no main effect of home stability on criminal activity for this age range, $F(2, 149) = 1.51$, $p = .22$, partial $\eta^2 = .02$.

Using the contracted data, which specifies if a participant lived in one home or had more than one placement, the findings were consistent. There was no difference in the current charges accrued and environmental stability between 8 and 12 years old between those in one placement ($M = 5.82$, $SD = 5.56$) and multiple placements ($M = 7.32$, $SD = 6.18$), $t(148) = -1.48$, $p = .14$. Likewise, there was no significant difference in total charges accrued between youth who lived in one home during this age range ($M = 14.06$, $SD = 11.16$) and those who lived in more than one ($M = 16.16$, $SD = 7.98$), $t(153) = -1.36$, $p = .18$.

4.6.1.3. Hypothesis 3.a.3. The data for hypothesis 3.a.3 were also analyzed using a one way ANOVA using the coding categories of 1 placement, 2 to 5 placements, and more than 5 placements. For the age range of 13 to 18 (or age at the time of assessment), there was no main effect of home stability on current criminal activity, $F(2, 151) = 2.86$, $p = .06$, partial $\eta^2 = .04$. When total charges were considered in the analysis, there was a significant main effect of home stability on criminal activity, $F(2, 153) = 7.51$, $p = .001$, partial $\eta^2 = .09$. There was not a significant difference in total criminal activity between participants who lived in one placement ($M = 12.39$, $SD = 10.25$) and those who lived in two to five homes ($M = 16.54$, $SD = 8.12$), $p = .06$. However, there was a significant difference between youths who lived in one home and those who lived in more than five ($M = 24.73$, $SD = 20.09$), $p = .001$; there was also a difference between those living in two to five and those living in more than five placements, $p = .03$. In both cases, a greater number of placements was related to more charges generated.

In contracted form, there was a significant difference between number of current charges accrued for those in one home from the ages of 13 and the time of the assessment ($M = 5.16$, $SD = 5.39$) and those in more than one home ($M = 7.41$, $SD = 6.11$), $t(148) = -2.10$, $p = .04$. Again, greater home disruption was related to more charges accrued. This finding remained consistent once total charges accrued were taken into account, with those living in one home consistently ($M = 12.58$, $SD = 10.49$) generating significantly fewer charges than participants placed in more than one home during this age range ($M = 16.50$, $SD = 8.58$), $t(153) = -2.40$, $p = .02$.

4.6.2. Hypothesis 3b

Hypothesis 3b predicted that home stability will be more related to criminal activity in participants with FASD given the high needs with which such individuals are known to present. Participants were rated as either having been in foster care or not and a point biserial correlation was run to determine if a relationship was present. It is an important question in this study, as it was found in this sample that a significantly greater number of participants in the FASD group were in foster care ($n = 65$) compared to the non FASD group ($n = 43$), $t(146) = 2.21$, $p = .03$. Related to this finding, a participant having ever been in foster care was statistically significantly related to both current charges, $r_{pb}(n = 183) = .17$, p (two tailed) = .02, and total charges accrued, $r_{pb}(n = 187) = .22$, p (two tailed) = .003. Thus it appears that having ever been in foster care may be slightly related to a having a higher number of charges, and a participant with a diagnosis of FASD was more likely to have been taken into care at some point.

The relationship between criminal charges and environmental stability for each age range was calculated for each group (with and without a diagnosis of FASD) and included in Table 15. The contracted data were used to simplify the analysis; participants were coded as either one placement or multiple placements. As with hypothesis 2.d., Fisher's Z was also calculated to assess for differences between the two groups.

From this table, it appears that only one relationship is significantly different between the two groups. Participants without a diagnosis of FASD evidenced a stronger relationship between environmental stability between ages 8 and 12 and charges accrued than participants with an FASD diagnosis. Apart from this, there were no significant differences in the relationships between stability and criminal activity detectable between diagnostic groups. However, from the data it appears that if anything, criminal behaviour by youth with FASD is *less* related to home stability than it is for those without an FASD diagnosis.

Table 15. Difference in Correlation Between Home Stability and Charges Accrued by Diagnostic Group

Hypothesized Relationship	FASD r (n = 84)	Non FASD r (n = 71)	Fisher's Z	Sig. (two- tailed)
Environmental Stability (0-7) and Current Charges	-.07	.22	-1.75	.08
Environmental Stability (0-7) and Total Charges	.12	.31	-1.22	.11
Environmental Stability (8-12) and Current Charges	.03	.22	-1.16	.25
Environmental Stability (8-12) and Total Charges	-.03	.29	-2.00	.05
Environmental Stability (13-18) and Current Charges	.07	.30	-1.43	.15
Environmental Stability (13-18) and Total Charges	.14	.25	-.70	.48

4.7. Hypothesis 4: Educational Assistance and Offending

The fourth hypothesis predicted that access to educational supports and resources would be related to both neuropsychological impairments and criminal activity.

4.7.1. Hypothesis 4a

It was expected that access to special educational resources (such as special education programs, resource room time, etc.) would be significantly related to more neuropsychological impairments. As there were only two categories (classified Help at School and No Help at School), a series of t-tests were performed on the neurocognitive measures to determine group

differences. As 15 t-tests were run, a Bonferonni correction placed the required probability level for significance at .003.

Overall, there were few differences between participants who had received assistance in school and those who had not on the neurocognitive measures of interest. There were no statistically significant differences between groups on nearly all of the assessment tools. However, there was a significant difference in ability to read words, $t(138) = -3.30, p = .001$, with those who received educational resources ($M = -1.45, SD = .90$) performing significantly more poorly than those who did not receive assistance in school ($M = -.94, SD = .87$). Other domains neared significant differences, such as Verbal Memory 2, $t(138) = -2.84, p = .005$, Attention 2, $t(138) = -2.26, p = .03$, and Receptive Vocabulary, $t(138) = -2.15, p = .03$; in each case, those who received educational assistance performed worse than participants who had not received help in school. Figure 6 displays the neurocognitive profile for each group as a graph.

4.7.1.2. Hypothesis 4b

It was anticipated that access to educational resources would have a moderating effect of criminal activity, based on the Streissguth et al. (1996) study which found that students who received educational resources were less involved in criminal activity. As such, it was hypothesized that neurological deficits left unaddressed would lead to more criminal offenses, and that participants who were provided with resources would have accumulated fewer offenses.

The relationship was first analyzed using a point biserial correlation. There was no significant correlation between educational assistance and current charges, $r_{pb}(n = 138) = -.04, p = .66$. Likewise, the relationship between assistance at school and total charges was insignificant, $r_{pb}(n = 140) = .04, p = .65$.

The data were also analyzed using a one-way ANOVA, with participants labeled as currently receiving educational resources, having received resources in the past, and never having received extra assistance in school. There was no significant main effect of educational assistance on the number of current charges, $F(2, 135) = .21, p = .81$. The findings for total number of charges was consistent with the correlational analyses, as there was again no main effect of educational assistance, $F(2, 137) = 1.55, p = .22$. From these analyses it appears that there was no relationship between offending and educational resources in this sample.

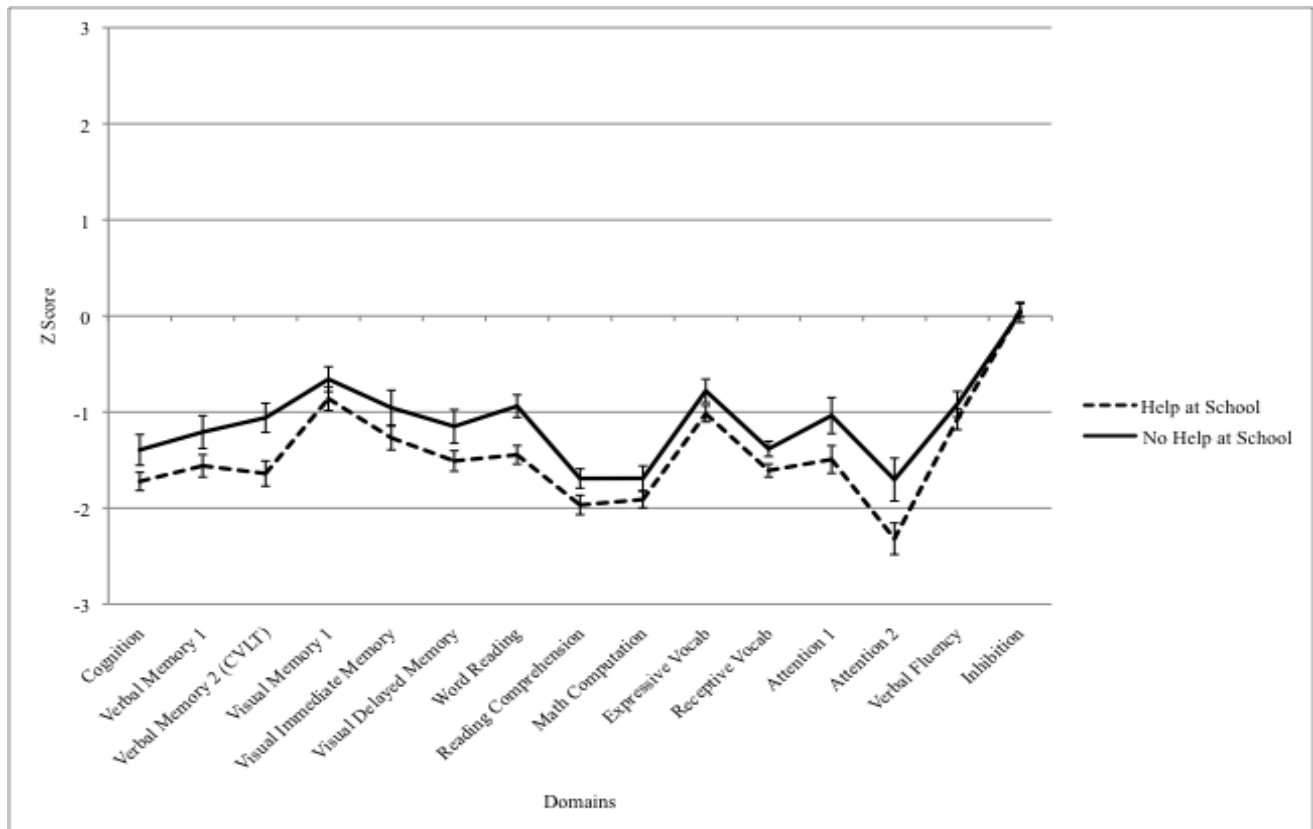


Figure 6. Mean scores on neurocognitive domains by assistance in school; the only significant difference at the $p < .05$ level was for word reading. Means are expressed in z score format; vertical lines depict standard errors of the mean.

4.8. Hypothesis 5: Substance Use and Offending

The fifth hypothesis anticipated that participants who admitted to substance use would have generated significantly more criminal charges than participants who had not.

4.8.1. Hypothesis 5a

It was expected that youth who acknowledged alcohol use would have accrued more charges than those who did not. Each was coded as currently using alcohol, not using alcohol, or having used alcohol in the past but currently clean. The data were analyzed using a one-way ANOVA. Regarding current charges, there was no significant difference in charges for those who admitted to using alcohol compared to those who did not, $F(2, 177) = 1.32, p = .27$, partial $\eta^2 = .02$. However, once total charges were taken into account, there was a small significant main effect of alcohol on number of charges, $F(2, 182) = 6.66, p = .002$, partial $\eta^2 = .07$. Those

who admitted to current alcohol use ($M = 16.98$, $SD = 10.00$) accumulated statistically significantly more charges than those who denied alcohol use entirely ($M = 9.18$, $SD = 8.73$), $p = .001$. Similarly, participants who acknowledged alcohol use in the past but were currently abstaining ($M = 16.75$, $SD = 10.22$) had accrued significantly more charges than those who have never used alcohol, $p = .01$. There was not a significant difference in number of total charges between participants who were currently using alcohol and those who were clean but had used in the past.

4.8.2. Hypothesis 5b

It was predicted that participants who admitted to marijuana use would have accrued more charges than those who did not. Each participant was coded as currently using marijuana, not using marijuana, or having used marijuana in the past but currently clean. The data were analyzed using a one-way ANOVA. Regarding current charges, Levine's Test revealed unequal variances between groups, $W(2, 177) = 4.01$, $p = .02$. This can be a problem for ANOVA when group sizes are unequal, as is the case in this analysis. However, the variance for the larger group ($n = 118$, $SD = 6.24$) is larger than the variance for those groups with smaller numbers ($n = 27$, $SD = 2.95$ and $n = 35$, $SD = 5.08$). Field (2009) noted that under these circumstances, the F value is likely to be conservative, thus we can be confident of any detected changes.

The analysis revealed a small significant main effect of marijuana use on number of current charges generated, $F(2, 177) = 3.60$, $p = .03$, partial $\eta^2 = .04$. Those who admitted to current marijuana use ($M = 7.52$, $SD = 6.24$) accumulated significantly more charges than those who denied ever using marijuana ($M = 4.33$, $SD = 2.95$), $p = .03$. There were no significant differences between participants who acknowledged current use and those who admitted past use ($M = 6.40$, $SD = 5.08$), nor was there any difference between youth who had previously used marijuana and those who had never used.

Similarly, there was found to be a significant main effect of marijuana use on the total number of charges accrued, $F(2, 180) = 9.13$, $p < .001$, partial $\eta^2 = .09$. Akin to the findings for current charges generated, current marijuana users ($M = 18.00$, $SD = 10.32$) accumulated significantly more total charges than those participants who had never engaged in marijuana use ($M = 9.35$, $SD = 9.08$), $p < .001$. There was no significant difference between youth who had used marijuana in the past but were currently abstaining ($M = 14.47$, $SD = 8.42$) and those who had never used marijuana. Likewise there was not a significant difference in total charges

between those who had acknowledged previous use and those who admitted they were currently using marijuana.

4.8.3. Hypothesis 5c

It was expected that youth who acknowledged other drug use would have accrued more charges than those who did not. Each participant was coded as currently using other drugs, not using other drugs, or having used other drugs in the past but currently clean. Substances that would fall under “other drugs” would include methamphetamines, LSD, psilocybin mushrooms, heroin, cocaine, ecstasy, prescription medications, etc. As participants engaged in this form of drug use less often, these substances were grouped together to maintain statistical power for analysis.

There was no main effect of other drug use on current number of charges accrued, $F(2, 173) = .50, p = .61$, partial $\eta^2 = .006$. When total charges were included in the analysis, there was found to be a small main effect of other drug use on number of charges, $F(2, 177) = 4.51, p = .01$, partial $\eta^2 = .05$. It was found that those who acknowledge current other drug use ($M = 19.04, SD = 10.51$) had accumulated significantly more charges than participants who denied ever engaging in other drug use ($M = 13.78, SD = 9.11, p = .03$). There was no significant difference found between those who had previously engaged in other drug use but were currently clean ($M = 17.65, SD = 11.54$) and youths who denied ever trying other drugs, nor was there a difference in total charges between those currently using other substances and those who had in the past.

4.8.4. Hypothesis 5d

It was also predicted that the relationship between substances and crime would be greater for participants with FASD, given their increased risk for substance use and abuse. Data were simplified to reflect if the participant had used a substance or not (the third category of previous use was eliminated to simplify interpretation). The relationship between criminal charges and substance use was calculated for each group (those with and without a diagnosis of FASD) and included in Table 16. As with earlier hypotheses, Fisher’s Z was calculated to assess for differences between the two groups.

From this table, we can deduce that the relationship between alcohol use and total criminal activity is stronger for participants with a diagnosis of FASD than it is for participants without a diagnosis. While the remaining relationships remain insignificantly different, it appears

Table 16. Difference in Correlation Between Substance Use and Charges Accrued by
Diagnostic Group

Hypothesized Relationship	FASD r (n = 78)	Non FASD r (n = 62)	Fisher's Z	Sig. (two- tailed)
Alcohol Use and Current Charges	.16	.00	.092	.36
Alcohol Use and Total Charges	.37**	.05	1.94	.05
Marijuana Use and Current Charges	.26*	.08	1.06	.29
Marijuana Use and Total Charges	.41**	.20	1.34	.18
Other Drug Use and Current Charges	.11	-.03	.800	.42
Other Drug Use and Total Charges	.23*	.07	.94	.35

* $p < .05$ ** $p < .001$

that overall, substance use is related to more charges for individuals with FASD than for those without.

5. DISCUSSION

While it was expected that young offenders with FASD would evidence a distinct pattern of neurocognitive deficits when compared to their peers without FASD, this was not borne out in the data. There was no difference in the neurocognitive profile between the two groups, indicating that in this sample, young offenders with and without FASD were dealing with similar profile of impairments. However, those participants diagnosed with FASD scored statistically significantly lower across the neurocognitive measures than young offenders without FASD; that is, the severity of their impairments was greater. It is important in interpreting these results not to consider these findings to be indicative of the entire young offending population, as the sample consisted of a disproportionate number of behaviourally challenged youth with a myriad of potential etiologies for their difficulties. Keeping this caveat in mind, these results suggest that differences between difficult-to-manage young offenders with and without FASD may not be due to differential abilities but rather differential severity of impairment, with young offenders with FASD demonstrating more serious deficits than young offenders without FASD.

Consistent with recent literature (Burd et al., 2010; Green et al., 2009; Mattson et al., 1999; Nardelli et al., 2011; Nash et al., 2013; Schonfeld et al., 2006), there was no significant difference in severity or profile on the neurocognitive measures for the sub-diagnoses of FASD in this sample. This suggests that amongst those included in this study, impairment may not be more severe for those with FAS when compared to what are considered the less serious diagnoses, pFAS and ARND. This is not surprising as the difference in the diagnosis, FAS versus pFAS versus ARND, is based on physical manifestations of the syndrome (the presence or absence of growth and facial features), whereas all three diagnostic categories require findings of central nervous system impairments to the same extent. These data suggest that CNS impairments may be similar, regardless of the presence or absence of physical manifestations of the syndrome, and that it is the common CNS findings that lead to problems, including criminal behavior. However, it is important to note that the lack of statistically significant differences does not indicate equivalency (Nickerson, 2000) as there are a number of potential confounding factors that could impact this finding.

That statistical differences were not found could have important implications, as children and youth diagnosed with FAS are commonly believed to be eligible for more services than those with pFAS or ARND, even though the severity of their behavioral impairments may not be

categorically different. These data suggest that there is no justification for limited behavioral services such as special education and alternative programming to and dispositions for individuals with FAS rather than with pFAS or ARND. However, it should be noted that recent research indicates families with children who are prenatally exposed have been able to access services without a formal FAS diagnosis (Astley, 2010), so this assumption may be erroneous and based on the general difficulty in accessing services for FASD.

More importantly in the context of this study, the justice system will likely view the youth differently when diagnosed with what is considered the “full” disorder. Given the confusion clinicians with mental health training have in understanding FASD (Wedding et al., 2007), it is likely that legal professionals familiar with clients with FASD will encounter difficulty regarding FASD awareness and knowledge in judges and other legal professionals. For instance, judges may only be familiar with the term FAS or FASD, without being aware of the spectrum that the diagnosis implies; youth with ARND would not be recognized to them as falling under the FASD label (Paley & Auerbach, 2010). Further, often the sole criteria for determining impairment in court is the IQ score (Brown et al., 2008) which does not adequately capture the impairments found in people with FASD, who may have serious impairment in some neurocognitive areas and/or adaptive behavior without a deficient IQ (Carr et al., 2010; Coles et al., 2010).

Additionally, judges and other court professionals may be unaware that the “lesser” forms of the disorder still require at least three areas of significant impairment – that the primary difference is in physical features, not behavioural or cognitive impairment (Brown et al., 2010; Paley & Auerbach, 2010). Chudley and colleagues (2005) note that the term “partial” in pFAS does not indicate lesser severity as the brain impairments may be the same. There is research to suggest that the physical features of the disorder indicate greater brain damage, as prenatal development of the face is driven by the development of the brain and the facial features have been found to be reflective of the severity of brain anomalies (Chasnoff, Wells, Telford, Schmidt, & Messer, 2010; Yang, Roussotte, Kan, Sulik, Mattson, et al., 2012). Yet if neurocognitive impairment is present and is required to the same extent in order to receive any diagnosis of FASD, then the question of why we regard some diagnoses as less severe than others must be considered, particularly when the mounting evidence agrees that there are not large differences in severity of impairment by diagnosis (Burd et al., 2010; Chudley et al., 2005;

Green et al., 2009; Nardelli et al., 2013). Some authors argue that the research literature does not support the subcategories of pFAS and ARND, as they cannot be consistently classified, and so children and youth should be considered either alcohol-exposed or non-affected (Burd et al., 2003). This changing perception is reflected in the professional literature, as a recent consensus statement regarding ARND in primary health care reported that alterations in brain development that result in cognitive and behavioural problems can be present even when the child's face and brain appear to be structurally normal (ICCFASD, 2011).

However, the previous finding regarding the lack of differentiation between sub-diagnoses must be considered in the context of the statistical analyses run, for which one of the subsamples was too small. Thus, while this finding is consistent with much of the current literature on the distinction of sub-diagnoses, the confidence on which this finding stands is suspect in this particular sample. Further, determining significance is not an either/or process and the p value is selected somewhat arbitrarily as a cut off score (Nickerson, 2000). Not meeting the predetermined level of significance is not indicative of insignificance on its own; a p value of .11, while not considered significant by the standard test, does not suggest that there is no distinction between subgroups. Nickerson also points out that there are a number of possible explanations for failing to reject the null hypothesis beyond the null hypothesis being accurate, such as poorly designed experiments or inadequate manipulation of extraneous variables. Also important to remember is that some researchers *have* identified differences amongst the subdiagnoses, such as Carr and colleagues (2010), who found children with pFAS scored lower on a measure of ability than children with ARND. Chasnoff and colleagues (2010) found those participants who met the strictest criteria for FAS were more impaired on a measure of general intelligence and impaired in more neurocognitive areas overall than participants with pFAS or ARND; there were no identifiable differences on neurocognitive measures between pFAS and ARND. It appears that there is some lingering controversy over the usefulness of the subdiagnoses. While there is not a consensus on overall neurocognitive functioning, at the least it appears that executive functioning skills between subdiagnoses are comparably impaired (Chasnoff et al., 2010; Mattson et al., 1999; Nash et al., 2013; Schonfeld et al., 2006).

Another important factor when considering these results is the difference in white matter connectivity and improved myelination in adults when compared to adolescents (Asato, Terwilliger, Woo & Luna, 2010). Specifically, as they age, adolescents tend to benefit from an

increase in white matter connectivity and increased myelination of axons. This likely contributes to greater voluntary control of complex behaviour by assisting in connecting a widely distributed brain. Asato and colleagues determined that white matter continues to mature into adulthood; those areas that continued to mature were areas that connected the prefrontal and orbitofrontal cortices to subcortical sections. In addition, it appears that white matter connectivity may be related to improved performance on response inhibition tasks. Given the affected regions (those involved in reward processing, response inhibition, and planning behaviour), it is likely that adolescence is a vulnerable time for risk-taking behaviour and the development and/or worsening of psychopathology. However, in general, people with a diagnosis of FASD demonstrate lower overall brain matter volume (Astley et al., 2009) than their peers, with some evidence that white matter may be particularly affected (Lebel et al., 2008). These results also fit with the findings of the current literature that indicates executive functions, such as planning and response inhibition, are one of the most consistently impaired areas of functioning for people with FASD (Brown et al., 2010; Green et al., 2009, Vaurio et al., 2008). Individuals with FASD have demonstrated particular difficulty when dealing with “hot” executive function tasks, which involve the reward/punishment system (Kully-Martens et al., 2013). Thus while adolescents with regularly developing brains may be susceptible to risky behaviour and have poor cognitive control when compared to adults, their FASD-diagnosed peers are possibly at an even greater disadvantage.

Of the measures administered to participants, the youths struggled most on the *WCST*; 38% of those who began the task were unable to complete it due to inability to tolerate the frustration or grasp the concept. Of those who were capable of finishing the test, the results mimicked those of the overall profile analysis; there was no difference in profile between those with FASD and those without but there was a difference in severity, with youth with FASD scoring consistently lower than youth without FASD. Of the participants who could not complete the *WCST*, no differences were found in profile or severity, indicating that youth without FASD who were incapable of (or unwilling to) finish the *WCST* were not distinguishable from youth with FASD who also discontinued the task. On the other hand, youth without FASD who could complete the *WCST* performed better across the board than youth with FASD who could complete the task. The *WCST* most measures impulse control and the ability to inhibit impulsive responding, and concurrently a degree of self-monitoring. It appears that there was a subset of

youth without FASD in this sample who have a measure of impulse control and self-monitoring that the other groups lack, and this group was generally more cognitively intact.

There were few meaningful relationships between the measures of memory and the number of system generated charges. Total system charges were found to correlate slightly with verbal memory ($r = -.13$), as measured by the *CMS*, *WMS*, or *WRAML*. This finding is somewhat as expected, in that it makes theoretical sense for verbal memory to correlate more with system charges than visual memory; however, only one verbal measure was significantly associated with charges and that relationship is small. Some memory deficits have been identified in life course persistent young offenders, particularly visuo-spatial memory (Raine, Moffitt, Caspi, Loeber, Stouthamer-Loeber, et al., 2005), but memory impairments have not been extensively examined in the youth offending literature. From these results, it appears that broadly speaking, the relationship between system charges and memory impairment was minimal at best.

It was unexpected that systemic charges did not relate more to memory impairments, particularly verbal memory, given that remembering one's conditions is a real-life verbal memory task. There are a number of explanations for this lack of finding. First, it is possible that the measures of use have insufficient ecological validity and are thus not accurately assessing memory impairments in individuals. A measure can be said to have ecological validity when the conditions of the test are similar to the situation in the real world and so the same cognitive resources are required (Burgess, Alderman, Evans, Emslie, & Wilson, 1999). This possibility is lent credence by the fact that the one small relationship found was for the best-normed and validated of the memory measures used. It also must be considered that the relationship between offending and neurocognitive deficiency is more complex than can be captured by one set of correlational analyses. It is likely not as simple as a one-to-one impairment-to-offending ratio and a number of other variables apart from memory difficulties contribute to systemic breaches. For example, perhaps memory deficits are compensated for by youth workers, parents, and/or guardians who remind the youth of his/her conditions. Given their criminal charges, we can assume a fair amount of antisocial behaviour in this sample, which may contribute to breaching conditions. There may also be a degree of antisociality in the youth's parents (Farrington, 2011; Silberg, Maes, & Eaves, 2012), leading them to be less encouraging of their child abiding by the terms of a probation as they do not hold prosocial values. Additionally, the unique characteristics of this sample may have influenced the results and the reported findings could be an anomaly.

Finally, it must be considered that the factors that contribute to the generation of systemic charges may be multifaceted and memory impairment does not capture a significant amount of this multifactorial variance. It was hypothesized that there would be meaningful associations between executive functioning deficits and charges of sexual offending, based on past research findings (Joyal et al., 2007). This prediction was not supported in this sample, as there was only one meaningful relationships between having accrued a sexual charge and attention or inhibition. There was a minor relationship detected between Attention 1 and sexual charges, but as the sample of youth who had been charged with a sexual offense was small and several of these participants were more severely impaired than others, additional analyses were run to determine the ability of the Attention 1 score to classify participants as a sexual offender. These analyses reveal that the use of Attention 1 scores resulted in a moderate to large effect in identifying those with a sexual charge, albeit based on a very small sample size so replication of such findings will be important before conclusions are drawn. This finding, if genuine and not a product of the sample, indicates that youth who commit sexual offenses may have significant attentional difficulties and thus may require closer supervision and environmental controls than other youth. For instance, this may involve keeping these youth from high-risk scenarios for offending, such as placement in foster homes with younger children.

However, the question remains as to why there was only one significant relationship between executive functioning and sexual offending in this study when past research has established that people charged with a sexual offense are impaired in this area (Joyal et al., 2007). To date, this area has been understudied, with little literature to inform as to the nature of the differences. It may simply be that the sample of sexual offenders in this study was too small to detect any meaningful associations, or too impaired overall. Likewise, the level of impaired attention may be underestimated, as the most impaired participants (i.e., those who struggled most significantly with Attention 1) were not administered the second measure of attention. This could lead to an overestimation of their abilities as one could assume Attention 2 would be more impaired than Attention 1, given the increased complexity of the task. The lack of relationship between inhibition and sex offending may be obscured by the fact that only those who could tolerate completing the *WCST* were included and thus reflect a more overall cognitively intact sample than those who were unable to complete the task.

Further, the lack of relationship may indicate a greater problem with the ecological validity of the neurocognitive measures employed. Researchers have suggested that executive functioning deficits may not be as apparent in the laboratory setting as in the “real world”. Nash and colleagues (2013) hypothesized that they may not have found differences between children with and without FASD on measures of executive functioning because of the false environment of a laboratory setting, arguing that real life tasks that require executive functions are not isolated from other cognitive and social demands. From this statement, it can be deduced that studies employing “hot” EF might be more relevant to real-world research than studies evaluating “cool” EF, as “hot” EF tasks are intended to take into account emotion and motivation, and consider reward and punishment (Kerr & Zelazo, 2004). Thus, studies that employ “hot” EF measures may better tap into the deficits that are directly linked to offending.

Despite this complaint against ecological validity, some studies have found good ecological validity for executive functioning measures. Burgess and colleagues (1998) assessed nearly 300 participants on 10 neuropsychological measures of executive function and found high levels of consistency between the test results and family/caregiver reported difficulties. The authors do acknowledge, however, that the different executive functioning tools were assessing for different underlying cognitive processes, which may also partially account for the difference in findings in the literature.

In this case, past research that has found a link between sex offending and executive functioning has used similar measures of executive functioning to the current study. In a review of the literature to date, Joyal and colleagues (2007) identified the *COWAT*, the *Stroop* test, the *WCST*, and *TMT-B* as commonly used instruments that have previously distinguished between sex offenders and the general population regarding certain aspects of impulsivity, verbal fluency, and attention. The current study battery also contained the *COWAT*, *WCST*, and *TMT-B*; however, much of the current sample was not administered the *WCST* or the *TMT-B* due to cognitive incapacity to complete the tasks. It may be that this sample was fundamentally different than typical research with sex offenders, in that past participants were better able to tolerate the testing situation than the adolescents in this sample.

To date, studies have primarily focused on adult offenders (Hancock et al., 2010; Joyal et al., 2007); it is possible that adolescent offenders do not demonstrate the same impairments as adult offenders, as impulsivity is a common feature of adolescence (Luna, 2009; Romer, 2010).

In addition, the brain of an adolescent is categorically different from that of an adult, in that the adult brain has greater white matter connectivity, which likely contributes to greater control of complex behaviour (Asato et al., 2010). These important differences suggest that research conducted with adult populations cannot necessarily be generalized to youth populations. The limited work published on executive functioning in adolescent sexual offenders suggests few, if any, differences between young offenders with a sexual offense and their peers (Kelly, Richardson, Hunter, & Knapp, 2002). 30 male youth with a sexual charge were compared to 20 non-offending but socioeconomically similar controls on a number of measures of attention and executive function, including the *TMT*, the *Stroop Test*, *WCST*, and the *ROCF*. Kelly and colleagues determined that response speed on verbal and design fluency was the only distinguishing variable between the groups.

Further, past research has compared sexual offenders to the general population or other offenders and found them to be impaired in terms of executive functioning, but it has not directly observed the relationship between executive functioning deficits and the number of criminal charges accrued. Thus, while offenders charged with a sexual offense may have poorer executive functioning skills than the average individual or offender, this does not indicate that the deficits themselves lead to sexual offending. Alternatively, it is possible that executive functioning alone does not account for significant variance in these complex multifaceted behaviours.

In their review, Joyal and colleagues (2007) also note that differences between sex offenders, general offenders, and the non-offending public may be obscured by grouping sex offenders in one category. They identified people charged with molesting children as scoring significantly lower than people who had sexually assaulted adults, the latter group of which was often indistinguishable from the general population. In this study, no attempt was made to qualify the nature of the sexual offenses in recording charges and so the possibility cannot be explored with this data. At any rate, the sample of youth charged with a sexual offense was so small, it is unlikely that classifying the type of sexual offense would inform this debate; however, this is an important avenue to consider for future research looking into executive functions in people convicted of a sexual offense.

Unlike past literature (Greenfield & Valliant, 2007; Hancock et al. 2010; Miuri, 2009; Raaijmakers, Smidts, Sergenat, Maassen, Posthumus, et al., 2008; Zagar, Busch, Grove, Hughes, & Arbit, 2009), this research project did not find any significant relationships between violent

offending and executive functioning deficits. The one meaningful correlation found between impairment of executive functioning and violent offense charges was a negative relationship between the number of perseverative errors on the *WCST* and the number of total charges accumulated; thus, more errors on the *WCST* (and thus the poorer the inhibition), the fewer charges one is likely to generate. This is counter-intuitive, in that it would be expected that poorer inhibition skills would be tied to *more* charges. However, it is not surprising in this particular sample, given that many participants could not complete the *WCST* due to frustration and discontinued the task. The significant relationship in this case is based on those young offenders who could tolerate completion of the *WCST* and are thus likely more cognitively capable.

Again, it is important to consider why this sample demonstrated no link between executive functions and violent offending when the literature supports this relationship (Greenfield & Valliant, 2007; Hancock et al. 2010; Miuri, 2009; Raaijmakers et al., 2008; Zagar et al., 2009). Many of the possible answers are those previously noted as explanations for the lack of correlation between executive functioning and sex offending: poor ecological validity of the neurocognitive measures employed, need for examination of “hot” EF to better mimic real-world decision making, methodological problems in the literature base (such as differential definitions of executive functioning and differential inclusion of criminal history; Hancock et al., 2010), or a biased sample of youths who could complete the *WCST*. Additionally, past research in which the link between executive functioning and violent offending has been established have used different instruments with their participants. Hancock and colleagues administered subtests of the *D-KEFS*: the Tower, Verbal Fluency, Color-Word Interference and Sorting tests. While some of the current sample completed these measures, there were not enough participants to include in analysis. Raaijmakers and colleagues (2008) administered the Go/No-go task, the Digit Span subtest of the *WISC*, Shape School, a verbal fluency task of generating words to fit a general category, the *Object Classification Task for Children (OCTC)*, and the Day-Night task. Greenfield and Valliant (2007) used the Porteus Maze, a task that assesses for planning and mental alertness but not inhibition. Zagar and colleagues (2009) employed a measure generated by the author called the *Zagar Executive Function Checklist*, an assessment of functioning that relies on self-reported (or caregiver reported) executive function difficulty, much like the *BRIEF*. It is likely that the different measures assess for different aspects of executive functioning or

have differing levels of accuracy in assessing executive function, which could partially account for this lack of consistency.

Additionally, there is a need for more research on the role of executive functioning in violence in young offenders, who may have fundamentally different abilities from adult offenders (for example, impulsivity being a common trait in adolescence and different brain development). Some research has focused on aggression in young children and identified a link between childhood aggression and poor inhibitory capacity (Raaijmakers et al., 2008). Likewise, Miuri (2009) found Japanese male adolescents with violent conduct disorder were more impaired than their peers with nonviolent conduct disorder on the number of categories achieved on the Kelo version of the *WCST*. In the current sample, a significant number of participants were unable to complete this measure of inhibition; therefore, it is possible that had a greater number of participants been able to tolerate the *WCST*, a relationship between inhibition and violent offending may have been found. Zagar and colleagues (2009) were able to identify poor executive functioning as a risk factor for homicide in a sample of young offenders; however, the term “executive functions” is not clarified in this research and judgments about impairment are based on self-reported difficulties. Research on what factors contribute to violence in adolescent offenders that employ more objective measures of executive function will be required to understand this relationship, or lack thereof.

Important to consider for this research project is that the unexposed adolescent brain is categorically different from the adult brain in terms of white matter connectivity and myelination, which continue to develop into the youth’s early 20s (Asato et al., 2010). This suggests that as people age, their ability to inhibit responses and control complex behaviour improves. However, it appears that for individuals with FASD, there is evidence of decreased myelination when compared to their unexposed peers (Sowell, Mattson, Kan, Thompson, Riley, et al., 2008). In a sample of children and adolescents, participants with a diagnosis of FAS were found to have significantly reduced white matter volumes in the cerebrum and parietal lobes compared to an unexposed control group (Archibald, Fennema-Notestine, Gamst, Riley, Mattson, et al., 2001). Thus, it appears that prenatal exposure differentially affects white matter, with an alcohol-exposed brain having less white matter at early ages. This suggests that the brains of individuals with FASD mature later than their peers and it is not currently known if they ever mature to the capacities of their non-affected peers. To date, research on the

development of the brain of people with FASD into adulthood has been very limited. One study conducted by Eckstrand, Ding, Dodge, Cowan, Jacobson and colleagues (2012) examined brain structure in young adults with and without prenatal alcohol exposure and found no differences between groups regarding white matter volume. However, it should be noted that their sample size was small (20 participants in total), alcohol doses in utero were reported to be low-to-moderate, and the average age of participants (approximately 19 years old) is younger than the age at which white tissue connectivity and myelination are thought to be complete (Asato et al., 2010). These factors, and the lack of supplementary research data, indicate that there is much left to learn about white matter development in adults with FASD.

In general, there were few meaningful relationships between any of the neurocognitive measures and number of charges accrued. There was a small association between Attention 2 and current charges; as well, there was a moderate relationship between Math Computation and index charges generated. It is interesting that Math Computation was related to a greater number of charges more than other measures, but makes sense if math scores are a proxy for a number of other neurocognitive domains, such as short term memory, working memory, and reasoning. As researchers are starting to understand, often the presentation of academic impairments is a result of the interaction of a number of underlying neurocognitive problems, particularly regarding memory and math skills (Rasmussen & Bisanz, 2010). The deficits in math that may instead be reflecting underlying deficits could be contributing to criminal behaviour as many crimes committed by youth are poorly reasoned and impulsive. In addition, memory struggles may leave such youth unable to recall that punishment may follow criminal activity, given that punishments doled out by the courts often occur at a significantly later date than the criminal activity itself.

However, neither the relationship between charges and Attention 2 nor charges and Math Computation remained significant once total number of charges was taken into account. This is surprising, as one may intuitively expect an association between neurocognitive deficits and charges. Past research has associated criminal behaviour with impaired cognition (Frisell, Pawitan, & Langstrom, 2012; Hanlon, Rubin, Jensen, & Daoust, 2010; Levitt & Lochner, 2001, Tiihonene, Eronrn, & Hakola, 1993), executive dysfunction (Aharoni, Vincent, Harenski, Calhoun, & Sinnott-Armstrong, 2013; Dolan and Anderson, 2002; Hanlon et al., 2010), deficits in language (Piquero, 2001), and attentional impairments (Bergvall, Wessely, Forsman, & Hansen, 2001). Yet as with the literature on executive functioning deficits in sexual and violent

offenders, this previous research has not looked directly at the correlation between neurocognitive deficits and charges generated, instead focusing on identifying impairments in offenders when compared to the general public or other groups of interest. The lack of relationship between specific deficits and criminal activity likely reflects the many influencing factors that contribute to offending behaviour, indicating that we cannot directly predict criminal behaviour from observed deficits. Just as cognitive scores are too complicated to directly link to brain volume (Nardelli et al., 2011), it is likely too simplistic to expect a particular neurocognitive score to correlate with criminal activity. The relationship is probably complex and involves many variables both stemming from and contributing to neurological impairment.

It was predicted that the strength of the relationship between deficits and charges would be greater for the youth with FASD than those without, as it was originally hypothesized that the criminal behaviour of individuals with FASD was directly related to neurological impairment whereas young offenders without FASD were thought to be more influenced by environmental and situational factors. This prediction was not found to be true in this sample, suggesting that neurocognitive impairments may not contribute more to offending in youth with FASD than in those without FASD. From this finding, it can be deduced that the young offenders with FASD were not at greater risk than other behaviourally challenging youth for criminal behaviour because of their neurocognitive deficits directly.

One possible confounding factor to the lack of relationship between neurocognitive deficits and offending in samples of people with FASD may be the structure surrounding the experience (Brown et al., 2010). For instance, cognitive testing settings are structured, with clear, concrete rules. Individuals with FASD may perform better on these tasks in a testing environment than when tried in novel, high-stress situations due to their neurocognitive deficits. In the presence of fast-paced and unfamiliar situations with no predetermined structure or routine, people with FASD likely decompensate and are at greater risk for behaviour problems. Thus they may not demonstrate the same impairments in structured settings as they would in the community. There has been some evidence to suggest people with FASD perform poorly when compared to controls on tasks requiring the incorporation of emotion or motivation into the decision-making process. They have been found to display a flat and random learning curve on the *IGT* when compared to non-exposed peers, suggesting a reduced sensitivity to reward and

punishment (Kully-Martens et al., 2013). This may account for why neurocognitive deficits were not found to relate more to offending in individuals with FASD.

In addition and as previously mentioned, scientists have begun to understand the complexity of neurocognitive impairment and accumulate evidence that neurocognitive deficits often interact to create observable difficulties (Rasmussen et al., 2009; Rasmussen & Bisanz, 2010; Schonfeld et al., 2006). For instance, memory difficulties could be due to problems with memory encoding and retrieval, but other factors such as attention, executive function, and ability to generalize from one situation to another, likely influence memory ability (Rasmussen et al., 2009). Difficulties in mathematic performance may be more tied to problems with working memory in children with FASD than in healthy controls. Rasmussen and Bisanz (2010) compared 21 children diagnosed with FASD to 20 control children on the *Working Memory Test Battery for Children* and two mathematical subtests of the *WJ-III Test of Achievement*. They determined that the children with FASD scored lower on quantitative concepts than applied problems and poorer on some tasks of working memory, specifically measures of phonological working memory. In addition, the scores of math achievement were more frequently correlated with scores of working memory for children with FASD than for children without the diagnosis, with group differences reduced once working memory scores were held constant. As the interacting effects of neurocognitive functions receive greater attention, it becomes increasingly apparent that the lack of direct relationship between offending and impairment is likely a function of the numerous variables that influence both criminal behaviour and neurocognitive deficits.

One final factor to consider is that both the FASD group and the non-FASD group consisted entirely of young offenders, all of who were deemed as difficult to manage in some way. Had a comparison group of youth with no history of offending been compared to a random sample of general young offenders, the findings of the analyses between executive functioning and offending may have been quite different. Such research will be valuable in clarifying how and if neurocognitive impairments contribute to offending behaviour.

Another focus of the present study was to examine how a stable home environment may be related with criminal behaviour in youths. It was found that between the ages of birth and seven, environmental stability was related to offending behaviours. That is, those youth who had experienced more than one home early in life had accrued more charges than those who had

lived in one home during this period. It is not possible from the correlational data to infer causality, as it is also likely that a traumatic early childhood could lead to both greater instability and later criminal behaviour. It is difficult to determine the precise nature of the relationship due to the number of complicating variables that cannot be controlled in experimental science. However, it is clear that participants who remained in one home had generated fewer charges. This suggests that intensive efforts to support biological families of at-risk infants and young children, as opposed to rapid use of foster care, may help to prevent later social problems including criminal behavior. Some research has found that maltreated children who remain in their biological home are less likely to engage in criminal activity than their peers who become involved in the child welfare system (Ryan & Testa, 2005); however, this finding could also be a result of the most abused children and youth being taken into care and less maltreated children remaining in the home. This finding of home stability being related to fewer charges accrued also suggests that crime prevention strategies may be improved by long term efforts such as identification of high risk mothers prior to pregnancy or during pregnancy, coupled with intensive parenting supports in early childhood, rather than environmental interventions after offending behavior takes place. Further research into the causal relationship between environmental stability and criminal charges would be required to determine if such interventions as proposed here would be effective.

Inconsistent with Streissguth and colleagues (1997), there was not found to be a significant effect of home instability between the ages of eight and twelve. Streissguth and colleagues determined that this was the vulnerable age range for children with FASD, in which a good home environment provided the greatest protection against negative life outcomes. The difference in findings is likely due to a variety of factors, one of which is the different samples. The Streissguth sample was strictly children with FASD; this may not be a particularly sensitive age for non-PAE children. In addition, their sample for secondary disabilities consisted of 60% Caucasian children, whereas the current sample was nearly 80% Aboriginal. This is very important when we consider what is known about the overrepresentation of minorities in poverty situations (Chand, 2000; Chow, Jafee, & Snowden, 2003), particularly the overrepresentation of Aboriginals in poverty in Canada (Trocmé, Knoke, & Blackstock, 2004; Wilson & Macdonald, 2010). Aboriginal families are more likely to have their children taken into the foster care system

in Canada (Trocme et al., 2004). Thus, the two samples were likely exposed to different life experiences apart from home stability.

This contrary finding could also be due to differences between studies in how home stability was coded and the reliability of information. Streissguth and colleagues (1997) asked the caregivers of their participants to report home stability, whereas the current research had to rely on self-reported home disruptions in the absence of other psychological and legal reports. Especially for the earlier age periods, the participants in this study may not accurately remember or even know of all their home placements. Thus, the information provided to Streissguth and colleagues was possibly more reliable, although the present analyses were run on a dichotomous variable (one home versus multiple placements), which likely minimizes the error in data collection. Finally, Streissguth and colleagues looked much more in-depth at the home life for their participants, with a “good” home environment meeting at least ten of twelve criteria; criteria included structure, rules, consequences, lack of parental substance abuse or mental illness, and financial security. Most likely, the current analyses were not specific or detailed enough to replicate Streissguth’s finding, as the quality of home life was not considered in this study.

Youth who had experienced more than one living placement between the ages of 13 and 18 (or their age at the time of assessment) had generated more charges than those who remained in one home. In this instance, instability may be leading to greater criminal activity or greater criminal activity may be leading to more instability. That is, criminal behaviour may instigate social services investigations into the family, resulting in foster care placements; it may lead to custodial sentences, which were coded as a home disruption in this study. Again, a traumatic home life may spur on both home instability and criminal activity. Once a youth is in alternative care s/he may socialize with other offending youths and learn more about the lifestyle that surrounds criminal activity (Dishion, McCord, & Poulin, 1999; Ryan, Marshall, Herz, & Hernandez, 2008). Thus it is very possible that home instability is both a contributing factor to criminal behaviour and a resulting factor from criminal behaviour.

One result became very clear from the data, however: that ever having been in foster care was a significant risk factor for later criminal behaviour. As mentioned previously, it cannot be determined from this data whether the increased risk was a result of the home disruption itself or the environment that resulted in the child being taken into care; in actuality, it was likely an

interaction of the two. This is consistent with previous literature, which has found foster care placement (particularly multiple placements) to be related to an increased likelihood of adult criminality (Barth, Duncan, Hodorowicz, & Kum, 2010; Baskin & Sommers, 2011; Ryan & Testa, 2005). While this relationship is likely at least partially explained by initial maltreatment in the biological home, other factors also impact the correlation. One variable that influences future likelihood of crime in children who have been in foster care is a high risk for abuse and neglect in the child welfare system due to the improper screening of foster families, inadequate training of social workers, and the failure to regularly supervise foster care children (Marzick, 2007). This is not a criticism of the social work or child welfare profession so much as an acknowledgement that they do not have the resources and training required to appropriately attend to their duties. As a result, many children and youth who experience maltreatment at the hands of their biological families are taken into care by the government and placed in other abusive situations. Regardless of the good intentions the child welfare has in protecting children, removal from the family often results in further school disruption (because of moving needs), lower likelihood of high school graduation and post-secondary attendance, and feelings of pain, confusion, oppression, and powerlessness (Bruskas, 2008). This is not to argue that children should never be taken into care, but just that all efforts should initially be made to keep a child with their family of origin before removal is considered.

It is also important to take the previous research on home stability into account when considering the findings of this study, as there were significantly more youth with FASD who had been in foster care than youth without FASD. The rate of FAS in children in the foster care system has been found to be 10 to 15 times greater than in the general population (Astley, Stachowiak, Clarren, & Clausen, 2002). There is some evidence that children with FASD are more likely to be taken into care even when compared to non-exposed but behaviourally challenging children (Rasmussen et al., 2010). Some researchers advocate for screening for FASD in all children taken into the foster care system (Burd et al., 2010), as it has been estimated that 80% of children in the foster care system have prenatal exposure to alcohol (Dicker & Gordon, 2004).

Another initial prediction proposed that environmental stability would be more important for youth with FASD, as it was hypothesized that their increased needs would benefit more from consistency. Interestingly, the opposite was found, in that there appears to be a relationship

between home stability and criminal charges for youth without FASD but not for youth with FASD. Specifically, stability between the ages of 8 and 12 was more related to crime for participants without FASD than for youth with FASD. This is particularly interesting, as Streissguth and colleagues (1997) identified that in people with FASD, this age range demonstrated the greatest relationship between poor home characteristics and secondary disabilities. However, as noted previously, Streissguth and colleagues examined twelve different characteristics of a “good” home and did not simply look at home stability. It may also be that the youth in the current sample had encountered too many traumatic and/or disruptive experiences by age eight, so that the buffering effects of a good home could not undo the early effects of prenatal alcohol exposure, poverty, abuse, multiple placements, etc. There are likely many confounding variables that were not accounted for in this study that account for the differences in findings.

One’s first instinct may be to assume from this result that criminal activity in youth with FASD is more related to the primary deficits than the postnatal environment; however, this contradicts the earlier noted result that youth with FASD did not evidence unique neurocognitive impairment when compared to other challenging young offenders. It is possible that it is merely the severity of the impairment that leads to an increased risk for criminal behaviour from those diagnosed with FASD. As it was noted that significantly more youth with FASD were taken into foster care, it is also possible that there was a lack of variability in environmental stability for the participants with FASD in this study. That is, there were not enough youth with FASD who had come from stable home environments to detect a relationship in the data. Additionally, it must be considered that there may have been errors in data gathering or the finding may be an anomaly. What is certain from this data set is that a stable home environment was more related to charges generated for young offenders without FASD than youth with FASD.

Another possibility regarding the importance of home stability is that it may be valuable to consider FASD under a stress-diathesis model. From this perspective, children who are prenatally exposed to alcohol are born with particular vulnerabilities that may be expressed when raised in dysfunctional, neglectful, and/or abusive homes. This is different from conceptualizing deficits as primary and secondary, as the hypothesis would be that under proper care and assistance, even primary disabilities could be offset by an appropriate home environment. However, such a model would need to account for those individuals raised in loving and

supportive home environments who still demonstrate the difficulties associated with FASD. It is possible that what qualifies as a supportive environment for individuals with FASD is qualitatively different than what is typically considered a positive home environment, due to the specialized needs often present in this population. There may be additional factors required to provide the same support for people with FASD when compared to those without FASD. This question may be possible to answer in the future, as the specifics of an appropriate home environment for people with FASD become identified in the literature and such variables can be studied.

In order to assess the ability of the school system to identify children in need of help, differences on the neurocognitive measures between participants who had received help in school and those who had not were examined. Overall, there were not found to be many meaningful group differences. Of the areas identified as distinguishing between the groups, Word Reading demonstrated the greatest difference, with Attention 2, Receptive Vocabulary, and Verbal Memory 2 also representing distinction between those who received help in school and those who did not. All but one of the domains of distinction is verbally based, which is unsurprising given that verbal deficiencies are likely those more easily noted by teachers in the classroom.

Overall, those who received help in school scored lower than those who had not, but most often not statistically significantly so. Further, many participants who had not received assistance in school demonstrated serious impairments at levels similar to those who were receiving or had received help. The lack of overall group differences may be explained by poor ecological validity of the measures; that is, impairment identified by a neurocognitive test is not reflective of difficulties in real world settings, and vice versa. Also possible, and likely given what is known about restricted access to resources for educational assessments (Johnson, 2012; Martinez, 2012; Odden, 1999), is that the education system may be doing a poor job of screening and identifying youth with cognitive weaknesses. That is not to say that the education system is failing these youth intentionally or maliciously, but rather that the institutions are not provided with adequate resources to meet the needs of the many differently abled youth and children who are placed within the system. Studies show that the appropriate allocation of resources for assessment, and the subsequent use of said assessment by the school, is integral to increasing achievement in students (Johnson, 2012).

There was no relationship between having received educational assistance and criminal activity in this sample; this is in contrast to the work of Streissguth and colleagues (1997), which identified educational resources as having a moderating effect on negative life outcomes in their sample, including involvement with the law. This study was unable to replicate their findings. This could be due to differences in the sample or methodology, as Streissguth and colleagues studied a predominantly Caucasian sample of children and youth with FASD and this sample was largely Aboriginal and consisted of behaviourally challenged youth with and without FASD. In addition, Streissguth was not looking strictly at criminal activity but all secondary disabilities; it is possible that educational services moderate other negative life outcomes but have less effect on offending behaviour. Finally, it must be considered that different school and educational boards may have different ways of determining disability and allocating aid, which would likely impact the ability to replicate findings from one study's sample.

Regarding substance use, it was predicted that those who admitted to use would have generated more charges than youth who abstained. This prediction was borne out in the data, as acknowledging current alcohol, marijuana, or other drug use was associated with having accrued more charges. It also appeared that ever having consumed alcohol resulted in more criminal charges, whereas past marijuana and other drug use was not related to criminal behaviour. Thus it seems that alcohol use at any time in their past was linked to higher charge rates, whereas only youth who engaged in current marijuana or other drug use were likely to have more charges.

There are likely a number of complicated variables that contribute to the relationship between offending and substance use. It may be due to distressing life circumstances, such as poverty or trauma, both of which may lead to both criminal activity (Brown, Henggeler, Bordino, & Pickrel, 1999; Carrion & Steiner, 2000; Hsieh & Pugh, 1993; Parker & Pruitt, 2000; Renn, 2000) and substance use (Khan, Murray, & Barnes, 2002; Najavits, Runkel, Neuner, Frank, Thase et al., 2003; Simons, Ducette, Kirby, Stahler, & Shipley, 2003; Smyth & Kost, 1998). It is well established that alcohol use lowers inhibitions and can lead to more criminal charges (Lennings, Copeland, & Howard, 2003; Miller, Levy, Cohen, & Cox, 2006; Rossow, 2004; Timko, Moos, & Moos, 2009), with some evidence that alcohol abusers with the most severe drinking patterns are also more likely to commit crimes in order to ascertain alcohol (McMurrin & Cusens, 2005).

The relationship between marijuana and crime is less well established. The causal role of marijuana use in offending behaviour has not been examined. There is some evidence that early cannabis use is associated with property and violent crime in adolescence and young adulthood, with the greatest relationship with marijuana use for those who began smoking prior to the age of 15 (Fergusson, Horwood, & Swain-Campbell, 2002). Past research has identified high rates of marijuana use amongst offending populations (Spunt, Brownstein, Goldstein, Fendrich, & Liberty, 1995). Marijuana use was found to associate with an increase in partner related aggression in a meta-analysis of 96 studies (Moore, Stuart, Meehan, Rhatigan, Hellmuth, et al., 2008); however, a recent longitudinal study linking substance use to increased risk of violence in teen dating situations found that unlike alcohol and other hard drugs, marijuana use was unrelated to future violence (Temple, Shorey, Fite, Stuart, & Le, 2013). Many of the youth in this sample who chronically used marijuana stated that they self-medicated for anxiety by smoking marijuana, which is consistent with the literature that people who have been traumatized may be more likely to self-medicate with marijuana (Chu, 2012). It may be that again, stressful life circumstances lead to both marijuana use and an increased likelihood of crime. However, a number of participants acknowledged engaging in criminal activity in order to have funds to purchase marijuana so even if it was not directly linked to their charges, marijuana use was still contributing to participants' offending behaviour. Another factor that may account for increased criminal activity in those who use marijuana is that merely by accessing the substance, youth are coming into contact with the criminal world and being exposed to procriminal attitudes. As marijuana is an illegal substance, youth must interact with those involved in the criminal world in order to acquire the drug and thus may leave themselves vulnerable to the manipulation of such individuals or falling into the procriminal mindset through this association.

As other drugs were all grouped together to maintain statistical power, it is difficult to sort out the nature of the relationship between other substances and criminal behaviour in this sample. Heroin, cocaine, and benzodiazepine use are all highly related to acquisition crimes, with users more likely to commit crime to maintain their drug habit (Comiskey, Stapleton, & Kelly, 2012) and the highest rates of acquisitive crime found in crack cocaine users (Best, Sidwell, Gossop, Harris, & Strang, 2001). Treatment of cocaine and heroin misuse in an offending sample has been found to result in a decrease in criminal activity (Comiskey et al., 2012; Lobmann & Verthein, 2009; Ribeaud, 2004; Schwartz, Jaffe, O'Grady, Kinlock, Gordon,

et al., 2009). In a meta-analysis of 30 studies that examined the association between substance misuse and crime, Bennett, Holloway, and Farrington (2008) found a three to four fold increase in risk for criminal activity of all kinds for people who use drugs as opposed to those who do not; the greatest risk of offending was for people who used crack cocaine. Similarly, a meta-analysis of partner violence examined 96 studies and found that the strongest association between substance use and psychological, physical, and sexual aggression was for cocaine (Moore et al., 2008). High rates of cocaine use have been found in perpetrators of homicide (Tardiff, Marzuk, Leon, Hirsch, Stajic, et al., 1994). Methamphetamine use has been found to increase one's risk for violent offending when compared to heroin use (Darke, Torok, Kaye, Ross, & McKetin, 2010). A recent evaluation of criminal behaviour in offenders who use methamphetamines has found that in comparison to other drug users, meth users have longer criminal records and are more likely to engage in property crimes (Gizzi & Gerkin, 2010).

Also important to consider is the literature on brain development in adolescents with a history of significant substance use, as it is becoming increasingly understood that extensive substance use at young ages may directly impact the development of the brain. It appears that considerable use of alcohol and marijuana in an adolescent sample may be connected to reduced white matter quality in association fibre tracts with frontal connections (Bava, Jacobus, Thayer, & Tapert, 2013). It has been hypothesized that the white matter quality reductions seen in heavy alcohol and marijuana using adolescents may be indicative of atypical myelin maturation (Bava, Frank, McQueeney, Schweinsburg, Schweinsburg, et al., 2009). Thus, the possibility must be considered that substance use directly impacts cognitive functioning due to aberrant brain development and is not merely a byproduct of neurocognitive impairment already present in FASD. However, this matter is further complicated by the finding that the brains of youth at high risk for substance abuse may be structurally and functionally different from the brains of youth not at high risk for substance use (Silveri, Tzilos, & Yurgelun-Todd, 2008). It is difficult to tease apart whether brain differences between youth who use substances and those who do not were present before the substance use or if the substance use is directly responsible for changing brain development. Further research examining the brain functions of youth with and without FASD who also use marijuana and alcohol may be able to help further elucidate this relationship.

It was expected that participants with FASD would demonstrate a stronger relationship between substance use and crime than youth without FASD, given both the high rates of

substance use in offenders (Bowes et al., 2009; Fazel et al., 2006; Kraanen & Emmelkamp, 2011; Novak et al., 2007) and the high rates of addictions in adolescents with FASD (Baer et al., 1998; Denys et al., 2011; Fast & Conry, 2009). The data support this hypothesis to an extent. There was a significantly stronger relationship between alcohol use and criminal activity for participants with FASD when compared to participants without FASD. Over all the drug categories, the young offenders with a diagnosis demonstrated more of a relationship between substance use and crime than youth without FASD. One possible explanation for this finding is that the neurocognitive deficits present in individuals with FASD make them more vulnerable to the negative effects of substance use, such as lowered inhibition and poor judgment, and thus more likely to engage in crime. It is also possible that the sample with FASD had less home stability and more trauma in their pasts, leading them to be more likely to both abuse substances and commit crimes. The relationship between offending and marijuana use in youth with FASD was stronger than the relationship between offending and other substances, which is an unexpected finding given that marijuana is typically the substance least related to criminal activity in the literature. It may be that youth with FASD are more vulnerable to manipulation and coercion by the criminal element they access when they purchase marijuana due to their desire to please others, or more vulnerable to the adoption of procriminal values in such situations due to poor reasoning and judgment skills. Further, the relationship may be due to the brain damage done in utero, which leaves the youth more susceptible to the deleterious effects of marijuana.

In attempting to deduce the relationship between substance use and criminal behaviour, a number of factors must be taken into account. Drug use may be related to criminal involvement due to directly causing crime, involvement in crime may promote or encourage substance use, and/or both offending behaviour and substance use may be caused by a third variable (MacCoun, Kilmer, & Reuter, 2003). There is support in the literature for all of these explanations. Moreover, if substance use does directly cause crime, it may be due to the pharmacological effects of the drug, it may be a result of acquisition crime (that is, crimes committed to obtain funds to purchase substances), or the nature of the illegal transactions could be the primary contributing variable to criminal activity. Thus, the prohibition of substances may be both a response to and a causal antecedent to drug-related crime.

In general, it may be of use to consider the issue of FASD in the context of the Risk Need Responsivity (RNR) theoretical framework, as this is the current standard when conducting correctional work (Andrews, Bonta, & Wormith, 2011). The RNR model proposes that in order for treatment to effectively reduce the risk of recidivism, it must meet certain criteria: 1) individuals with the highest level of risk to reoffend receive the most and the most intensive treatment when compared to lower risk offenders; 2) treatment targets must be criminogenic needs; that is, needs that once addressed directly reduce the risk of reoffense (e.g., substance use, employment problems); and 3) treatment and interventions must take into account the unique skills, deficits, and characteristics of the individual. From this perspective, a diagnosis of FASD would impact treatment particularly at the needs and responsivity levels (while we might infer people with FASD to be high risk, it is not possible to speak to the level of risk for each individual and level of risk likely varies within the diagnosis of FASD). In particular, the primary deficits would best be conceptualized as a responsivity issue, in that people working with offenders with FASD will have to consider their difficulties with memory, abstract reasoning, generalizing between situations, and learning from standard methods of punishment and reinforcement in both case and treatment planning. The secondary deficits may then be conceptualized as tying into the needs category, in that substance use, employment difficulties, problematic peer relationships, and struggles in school are common both in people with FASD and in offending populations. By addressing the criminogenic needs of people with FASD in a responsive manner, the risk of offense/reoffense should correspondingly decrease.

5.1. Recommendations

Although a direct connection between neurocognitive impairment and the number or types of charges accrued was not found in this study, participants with FASD in this sample were dealing with greater impairment than participants without FASD. Recognizing the role of neurocognitive deficits in criminal activity is to recognize that behaviour can be linked to brain-based disabilities rather than intentional opposition (Burd et al., 2010; Kully-Marten et al., 2013). As the Correctional Service of Canada is dedicated to public safety above all other factors (Ogloff, 1995), FASD should be considered a mitigating condition during sentencing, and alternatives to incarceration should be considered, including therapy, programs based in the community, and other non-custodial measures (ABA, 2012).

Recently, some researchers have advocated for a change in how the justice system views culpability. They argue our justice system's current focus on criminal responsibility as an either/or concept is out-dated and unsupported by the neuropsychological data on impulse control, planning, judging, reasoning, and decision making (Mela & Luther, 2013). The literature supports the notion that one can have varying degrees of comprehension and control regarding one's criminal behaviour. As treatment and reduction of criminal behaviour must take into account an individual's level of mental accountability, criminal justice professionals struggle with how to place people whose actions cannot be discounted as entirely outside or within their control. For example, people with FASD show an impaired ability to make cause and effect connections (Boland et al., 1998) and have difficulty with abstract concepts such as time (Malbin, 2004; Mattson et al., 1999). These difficulties often lead to repeated offending and the worsening of their situation through breaches of parole and probation, such as failure to appear in court (Mela & Luther, 2013).

Mela and Luther (2013) argue for a possible verdict of *diminished* capacity for offenders with some form of cognitive impairment. Capacity for guilt requires four conditions: understanding, appreciation, reasoning, and choice. If any of these areas of functioning are deficient, one's responsibility for criminal actions must be questioned. Thus, capacity exists along a continuum. Many people with organic brain disorders, such as FASD, have difficulty with such functions as comprehending, abstraction, remembering, and analysis (Chasnoff et al., 2010; Green et al., 2009; Howell et al., 2006; Manji et al., 2009; Mattson et al., 1999; Olson et al., 1998; Schonfeld et al., 2006); these faculties are not related to the typical understanding of an "insanity" defense - that is, that one is disturbed in areas of perception and belief (exhibiting hallucinations or delusions). However, individuals with observable mental impairment, such as those people with Down's Syndrome, are rarely presented in the criminal justice system as the system recognizes that their criminal activity is related to their level of intellectual functioning (Mela & Luther, 2013; Paley & Auerbach, 2010). This suggests that the system realizes cognitive impairment does impact capacity; this realization has been yet to be extended to those without observable markers of impairment. Mela and Luther (2013) point out that complete and full understanding of an act is not the same as sufficient understanding. Their proposal for a possible verdict of diminished responsibility would recognize that *mens rea* (or "guilty mind") is existent but not complete, rendering the accused minimally responsible for the crime. People

with diminished responsibility could be diverted to the proper rehabilitative channels and strategies employed with individuals found Not Criminally Responsible could be similarly employed with this group. This approach to determining responsibility is also more in line with the criminal justice system's purported mission of public safety over punishment (Ogloff, 1995) as it may reduce recidivism rates amongst people unable to benefit from standard methods of punishment.

Of the many problems in tackling the issue of FASD in corrections is the lack of FASD programming available for adolescents and adults, both in the community and in institutions. Adults with FASD may have to qualify for another disorder in order to receive any resources or services, depending on whether provincial or state law recognizes FASD as a disability (CBC, 2007). The ABA (2012) recently recommended that law and policy makers recognize people with FASD as eligible for appropriate medical services and disability benefits in hopes of reducing their involvement with the criminal justice system. In 2011, there were no Correctional Service of Canada (CSC) programs for FASD or any other mental disorder resulting in impaired cognition; all CSC programming was taught at a Grade 9 level (Dej, 2011). This indicates a serious need to accommodate programming to include lower functioning offenders of all maladies and disorders, not only those with FASD. Burd and colleagues (2010) outline specific strategies in planning programming for offenders with FASD, such as using small groups, short directions, and longer duration of programming.

Particularly when it comes to youth, it is recommended that FASD screening be incorporated into the justice system (ABA, 2012; Bisgard, Fisher, Adubato, & Louis, 2010; Paley & Auerbach, 2010). While screening cannot be used as a substitute for a complete diagnostic assessment, early screening decreases the risk of developing secondary disabilities, such as criminal involvement (Burd et al., 2010). Screening can be used to identify indications of FASD for children and youth who can then be sent for a thorough assessment (Bisgard et al., 2010). Thus ideally, screening would occur in populations known to be at risk for PAE, including children of mothers in a substance abuse program in prison, children in the foster care system, and siblings of children diagnosed with FASD. As FASD often goes undiagnosed, particularly in the absence of the physical features (Chudley et al., 2007; Dubovsky, 2008), the criminal justice system may be the first opportunity for screening. The Canadian Correctional system routinely screens for mental health and substance use disorders (Burd et al., 2010);

including a screening measure for FASD would increase the thoroughness and likely the accuracy of the intake assessment. In a recent survey of probation workers in British Columbia, it was found that 60% of youth at-risk for FASD would have gone unrecognized if their standard screening tool had not included questions specifically querying for FASD (Bisgard et al., 2010).

Screening measures for offenders with FASD were not available at the time of this writing, but several were in development and showing promise. Goh, Chudley, Clarren, Koren, Orrbine, et al. (2008) advocate using the Asante Centre for Fetal Alcohol Syndrome Probation Officer Screening and Referral Form as a screening tool in youth justice. The Asante Centre has been comparatively effective in trying to classify youth at risk for FASD (Bisgard et al., 2010). Goh and colleagues (2008) note the Asante instrument assesses for social, personal, and mental health factors. Currently a project in Colorado is being implemented with the goals of referring children for possible diagnosis, involving family members and other influential people in the child's life to contribute to the individualized case plan for each youth, and to track and monitor the success of the system (ABA, 2012). Brown and colleagues (2010) reported some success by using the brief screening tool of the FASD Youth Justice Project in Manitoba, noting that it is not in general use outside of the province; details of the tool's success were not provided. It is too preliminary for results at this stage, but such ventures lend optimism that increased resources, knowledge, and understanding of FASD will lead to strategies for management. Burd and colleagues (2010) recommend that everyone entering the criminal justice system be screened for FASD.

There are limitations to screening measures for FASD in a juvenile population. For instance, while researchers are working on screening tools, validation of such measures has been a problem (Bisgard et al., 2010). Often records of maternal drinking are unavailable or unreliable. Further, there may be resistance from the offenders to be screened for FASD as many see it as a stigmatized label.

Currently, it appears that prevention of FASD is still the most cost-effective and compassionate method of combating alcohol-related disorders within the justice system. Prevention can be difficult; many of the mothers who drink while pregnant are dealing with substance abuse issues (Astley et al., 2000) and there are few substance abuse treatment programs for pregnant women. Fortunately, research on some preventative efforts has yielded positive results, as is the case for the First Steps Program run out of Edmonton (Rasmussen,

Kully-Martens, Denys, Badry, Henneveld, et al., 2012). The First Steps program is based on Washington state's Parent-Child Assistance Program (PCAP), with the central aim of preventing future drug and alcohol exposed births in mothers at high-risk for substance abuse during pregnancy. It is based on an advocacy/case management model, which offers maternal support for up to three years. A study of the program conducted through the University of Alberta found that in a sample of 70 at-risk mothers, there was an increase in the consistent application of family planning methods (from 36% to 56%), as well as a meaningful drop in the use of welfare (from 92% to 72%). Further, 44% of participants were abstinent from drug use by the study's conclusion and 35% were abstinent from alcohol use. There was a significant reduction in identified needs, with 17 of 20 categories demonstrating decline; the largest reductions were found in Independent and Financial Issues, Difficulties with Community Resources, Addictions Problems, Health Issues, Social Problems, and Transportation Difficulties. There was also a meaningful increase in the number of reported goals for participants, with the categories of Parenting, Self-Care and Health, and Resources showing the largest increase.

On a larger scale, there has been some success with the Early Start substance abuse program as well. In a retrospective cohort study consisting of 49, 985 participants, mothers who were screened for substance abuse and treated while pregnant had significantly lower rates of neonatal assisted ventilation, preterm delivery, and low birth weight than participants who were screened but refused treatment (Goler, Armstrong, Taillac, & Osejo, 2008). Even women who were screened and assessed but not treated had better neonatal outcomes than women who were just screened, as the assessment component of the program consists of education as well. Women with substance abuse who were treated did not have higher rates of intrauterine fetal death (IUFD) compared to abstaining controls, but participants with substance abuse who were not treated had significantly greater incidence of IUFD compared to both controls and substance abusing participants who received treatment. This is not due to greater substance use by those who refused treatment, as the screened and treated group had a more severe pattern of substance use than those who refused assistance. It is possible those who accepted treatment were more motivated to seek help or they may have been more aware of the risks due to familial and friendly pressure.

A cost-benefit analysis of the Early Start program revealed that participation in the program led to lower overall costs by an extent that is substantially more than the cost of the

program itself (Goler, Armstrong, Osejo, Hung, Haimowitz, et al., 2012). Using costs as determined by the Kaiser Institute's Cost Management Information System, the authors estimated that the annual savings per four million births would be approximately \$2 billion. The most notable costs were those related to preterm birth, which per infant cost 2.9 times more in the screened only group than in the screened and treated group. At the same time, maternal and neonatal outcomes are likely improved (Goler et al., 2008). Rasmussen and colleagues (2012) argue that the lack of social costs incurred with prevention of FASD demonstrates the cost-effectiveness of substance use interventions with pregnant women. Thus it seems that prevention and assistance as provided by substance abuse treatment to pregnant women is not only the compassionate, caring response, but also justifiable economically.

5.2 Limitations

While the use of real-world collected data has advantages, such as greater external validity and generalizability (Barab & Squire, 2004; Jensen, Hoagwood, & Trickett, 1999), it also presents the current research with a number of methodological problems. While I recognize that socioeconomic status is greatly related to criminal behaviour (Ellis & McDonald, 2001; Hsieh & Pugh, 1993; Wikstrom, 1991), and it would thus be valuable to consider SES as well as age, ethnicity, and gender, that information was not collected at the time of assessment and so cannot be included. In addition, the sample sizes for the groups with and without FASD were unequal, which reduced the power of the statistical analyses run.

It is also important to keep in mind that some of the youth in the sample may have been on medication. This was not queried at the time of assessment, unfortunately, and so cannot be considered in this data set. However, the effects of unknown medication may have confounded the results, if some participants performed better on some measures than they otherwise would have due to medically improved mental faculties.

Moreover, while every participant diagnosed with FASD was exposed to alcohol in utero, it cannot be certain that they were not also exposed to other substances known to affect neurodevelopment. Many women who abuse alcohol while pregnant are polysubstance users (Goler et al., 2008; Moyer, 2013). This information was queried at the time of diagnostic assessment, but is questionable in the same way maternal drinking reports are suspect; that is, fear of stigma may have led to denial of prenatal substance use, the mother may have had impaired memory due to substance use, or the information may not be available due to the youth

being in foster or adoptive care. This is a problem that also affects our control group, although it is less likely that women who abstained from alcohol during pregnancy engaged in prenatal drug use.

Another concern is the nature of the comparison group. Prenatal alcohol exposure is queried in the standard referral procedure, in which the courts and youth court workers examine an offender's life history before deciding on the type of assessment required. From this, I can be confident that the control group was not prenatally exposed to alcohol, but I cannot be entirely certain. As discussed previously, there are a number of barriers to a determining prenatal exposure, such as unavailable or incomplete birth and medical records for adopted and fostered children (Chudely et al., 2005, 2007; May et al., 2004), and the mother's denial due to shame or damaged memory, which is particularly a problem in retrospective research studies (Astley, 2006; Chudley et al., 2005). However, the vast majority of youth seen were born in Saskatchewan after the commencement of FASD awareness campaigns, so it can be assumed that most Saskatchewan women of child-bearing age were aware of the dangers of drinking while pregnant.

The control group presents another potential problem, in that they were referred by the court to have a neuropsychological evaluation completed. As such, I can infer that they have some sort of behaviour dysfunction that makes them difficult for the courts to manage. Because of this, I cannot assume that they are representative of the offending population as a whole, as many youth go through diversion (that is, receive community service or probational conditions) and are only seen by the court once. The lack of a pure control group is a common issue in FASD research, as most participants are recruited through some form of assistance or medical program, which is likely to see those youth with the greatest behavioural and cognitive difficulties, regardless of etiology (Nash et al., 2013; Rasmussen et al., 2010). That the problem is common does not eradicate the issue in this sample; both the youth with FASD and without are likely to be more chronic offenders than the general criminal population. I therefore cannot make assertions as to the nature of the differences between offenders with FASD and the entire criminal population. However, this research was able to determine if young offenders with FASD demonstrate a distinct pattern of deficits when compared to other behaviourally impaired young offenders.

Another issue with the current research design is that much of the sociocultural information was necessarily gathered through participant self-report. This is of a concern because I cannot be sure of the accuracy of such information; some youths may intentionally misreport to minimize or exaggerate their troubles, whereas others may not have accurate knowledge of their histories. As much as possible, information was verified with independent sources, such as previously completed assessments and the pre-sentencing report. However, for some individuals, little additional documentation was provided, leaving their self-reports as our sole source of information.

The possibility of criterion contamination is also a concern. Criterion contamination is the result when both the independent and dependent variables are too similar in content (Lamberty & Heilbrunner, 2003), which nearly assures the null hypothesis will be rejected. In the case of the proposed research design, this becomes an issue because diagnosis of an FASD is predicated on neuropsychological impairments indicated by the measures of interest. I run the risk of tautological reasoning whereby my experimental groups are distinguished by differences on the tests, which I then propose to examine for differences. However, the design has several safeguards in place to attempt to minimize contamination. For instance, diagnoses were not based on the neuropsychological data alone; this information was taken together with physical measurements and a history of PAE, after which a medical specialist made the diagnoses. While each participant assigned a diagnosis of an FASD evidenced neuropsychological deficits, categorization between FASD subdiagnoses was largely determined through the physical examination. In addition, the majority of the control participants also demonstrated cognitive deficits, eliminating concerns that such impairments would distinguish them from the experimental groups. The central research question in this study is *where* do the differences lie? Criterion contamination should not be of concern in detecting such differences, assuming differences exist.

Another methodological concern arises from the nature of the assessment battery used. The battery was not uniform across all individuals (as mentioned, some were tested with different, age-appropriate measures, and others received the newer version of tests as the tests were released). This was clinically important when the data were collected, as it was vital to be able to reference the proper norms and use the most current products for each assessment. Unfortunately though, the difference in battery makes direct comparisons between measures and

subtests somewhat questionable. In all possible cases, I have provided the research data on the correlational studies completed on each instrument; often, this provides a direct intercorrelation estimate for each measure used. All interpretation of results was done with these relationships in mind. Although this is far from a perfect solution to the problem, these are the kinds of problems that arise when conducting research in real-world settings.

A possible confounding variable is related to the number of current charges the youth generated compared to total (including past) charges. Typically, a person is charged with more crimes than the number of which they are eventually convicted. Thus, associating initial charges (which are likely to be many) with past convictions (which are likely to be fewer) is not ideal in determining the relationship between the number of charges accrued and other psychosocial and neurocognitive variables. It was unavoidable in this case, as many of the participants in this study had yet to be sentenced (the youth were referred for an assessment to help determine sentencing for their charge[s]) and the full number of initial charges for past convictions was not known. However, the link between past and current crimes would be more valid if the sample had been sentenced and thus the number of current convictions instead of charges was known.

Related to this limitation of past and current crimes is that a large amount of the criminal activity for this sample was unknown. Past convictions were not provided for all participants, which renders the criminal history for many participants unclear. In addition, offenders are often not arrested and charged for all of their criminal behaviour, indicating that they may have engaged in more criminal behaviour than that for which they were charged. This is important missing data that could inform much as to the actual relationship between offending and psychosocial and neurocognitive factors. However, this is a common problem in forensic research, as rarely is the true extent of criminal behaviour known to researchers.

5.3 Future Research

The goal of the present study was to hopefully inform strategies to reduce the likelihood of reoffending in youth with FASD. As unique deficits were not discovered in this sample, it is not possible to identify how strategies should be adapted. However, as mentioned, this sample was not representative of the young offending population as a whole; the control group was a subset of behaviourally challenged young offenders who may be more similar to their peers with FASD than the average young offender. The field would benefit from research that assesses neurocognitive and social differences in a more general young offending sample (i.e., each youth

in this sample was referred for neuropsychological assessment because of suspected cognitive problems and cannot be considered typical of young offenders).

It was not possible from this sample to determine how much variance in offending could be accounted for by environmental situations alone as opposed to the interaction of neurocognitive impairment and their environment. This distinction will be important to make, as it could help inform strategies to minimize risk for criminal behaviour preemptively; that is, do children and youth with FASD require different environments than children and youth without FASD to be successful? Or is it merely the increased number of children with FASD with disrupted or traumatic social histories that leads to the increased risk for offending?

While identifying areas in need of intervention is important, it is also vital to determine whether the current strategies used to treat specific problems are relevant to youth living with FASD. More studies are required that examine whether the evidence-based interventions used successfully with children who do not have FASD, but who exhibit similar impairments and problems, are also capable of success with children and youth with FASD (ICCFASD, 2011). Interventions can then be modified if they are found lacking.

Researchers remain hopeful that there may be critical periods in the postnatal environment during which the teratogenic damage done to the brain during prenatal exposure to alcohol can be ameliorated by enrichment (Berman & Hannigan, 2000). That is the basis for recent research at the Hospital for Sick Children in Toronto that looks into the possibility of capitalizing on neuroplasticity and improving brain function in individuals with FASD (McIlroy, 2011); the idea is to focus on increasing self-regulation, emotional processing, impulse control, and social understanding. According to the current animal literature, scientists are having some postnatal success in reducing the deficits of prenatal alcohol exposure. Monk, Leslie, and Thomas (2012) determined that the teratogenic effects of alcohol in the hippocampal system of rats may be improved by perinatal choline supplements. It is hypothesized that motor hyperactivity attributed to prenatal alcohol exposure in rats might be attenuated by the stimulation of hypothalamic orexin synthesizing neurons, given the role such neurons play in a test of homing responses (Stettner, Kubin, & Volgin, 2011). In a review of the animal literature on prenatal exposure to alcohol, Kelly, Goodlett, and Hannigan (2009b) concluded that the neurobehavioural sequelae present in prenatally exposed rats could be ameliorated by such actions as neonatal handling, physical exercise, exposure to more objects, greater opportunity for

social interaction, and motor training. This data lends hope to the potential discovery of interventions to aid people with FASD in managing their symptoms. Researchers are careful to note, however, that while animals studies can greatly inform the profession's understanding of deficits and hopefully indicate potential avenues for intervention, results from animals studies cannot be taken as indicative of similar results in human populations (Berman & Hannigan, 2000; Kelly et al., 2009b). It is possible that such hypothesized interventions will have an effect on ameliorating neurocognitive damage in people but such treatments will have to be tested on human participants before conclusions can be drawn.

On a more general note, there is greater need for studies that examine the breadth of development in FASD (ICCFASD, 2011). It is important that families and professionals are aware of the difficult transitions that can arise for the child with FASD in becoming an adolescent, or an adolescent becoming an adult.

It will be useful for FASD prevention to examine in greater detail the differences between mothers who choose to drink while pregnant and those who abstain. To date, it appears that women who acknowledge moderate to heavy prenatal alcohol use are more likely to smoke, use other drugs, and be physically abusive to their children (Moyer, 2013). These are all factors that would likely worsen the child's prognosis independent of exposure to alcohol, and thus must be teased from the equation in order to understand the relationship between FASD and crime. Additionally, research on the genetics of parents who produce offspring with FASD could shed light on any hereditary components of the disorder.

Past research on the role of social functioning in people with FASD indicates that the presence of social difficulties in people diagnosed with the disorder is likely not strictly a result of neurocognitive impairment (Mattson & Riley, 2000), and that children lacking in social confidence often experience rejection from their peers, victimization, and demonstrate greater incidence of problem behaviours (such as aggression and poor behaviour control; Welsh & Bierman, 2001). Thus the social impairments seen in individuals with FASD may contribute in a meaningful way to the higher risk of criminal behaviour in this population. This research did not include a measure of social skills or functioning and so could not address this issue. However, as the neurocognitive deficits in FASD were not found to directly relate to criminal charges generated in this sample, examining the relationship between social ability and offending would be a valuable avenue of research.

It has been speculated that social functioning may be related to “hot” EF (Hongwanishkul, Happaney, Lee, & Zelazo, 2010). Research that incorporates an element of reward and punishment for executive function tasks may better access the deficits seen in people with FASD by accounting for emotion and motivation. These tasks are likely more representative of the decision-making process for criminal behaviour, which is possibly characterized by heightened emotion and typically reward motivated. Thus research into the relationship between “hot” EF and criminal behaviour in people with FASD would be valuable to further our understanding of why people with FASD are more likely to be involved in offending behaviour.

Another area of functioning not evaluated in the current study was adaptive behaviour. It may be that the neurocognitive impairment noted in individuals with FASD does not directly lead to greater criminal activity; instead, these deficits lead to impaired social awareness, poor ability to read emotions, and difficulty adapting to unfamiliar situations, which in turn render people with FASD more vulnerable to manipulation and less aware of how their actions impact others. People with FASD have been found to be significantly more socially inept than their peers (Greenbaum et al., 2009; Nash et al., 2013; Schofeld et al., 2006), even after behaviour problems are held constant (Rasmussen et al., 2010). Further, past research indicates that individuals with FASD have significant problems with adaptive behaviour when compared to non-PAE children and youth (Jirikowic et al., 2008; Streissguth et al., 2004). Deficits in adaptive behaviour are often significantly greater than would be expected given IQ scores (Streissguth et al., 2004); in fact, adaptive behaviour may be most impaired for people with ARND, who have been found to score lower than people with FAS and pFAS (Carr et al., 2010). It would be valuable to determine the relationship between adaptive behaviour, social functioning, and criminal behaviour in people with FASD to further elucidate the connection between people with FASD and an increased risk of offending behaviour.

As the data for substance use were correlational in nature, it was not possible to determine the causal role of substance use in criminal activity. Future research may focus on determining timelines of substance use to evaluate whether the use of substances co-occurs with much criminal behaviour, perhaps due to greater general distress or the antisocial lifestyle, or if using substances is causally related to future offending in youth, both with and without a diagnosis of FASD.

6. CONCLUSION

People with FASD have demonstrated neurocognitive impairments directly tied to prenatal damage in the brain, such as brain structure and neurological deficits (Astley et al., 2009; Coles et al., 2011; Franklin et al., 2008; Green et al., 2009; Jacobsen et al., 2008; Kalberg et al., 2006; Nardelli et al., 2011; O'Brien et al., 2012; Stephen et al., 2012), cognition (Carr et al., 2010; Chasnoff et al., 2010; Howell et al., 2006; McGee et al., 2009; Nash et al., 2013), receptive and expressive language (McGee et al., 2009; Nash et al., 2013), achievement in school (Howell et al., 2006; Olson et al., 1998; Streissguth et al., 2004), memory (Kully-Martens et al., 2012; Manji et al., 2009; Mattson & Roebuck, 2002; Olson et al., 1998; Pei et al., 2013), executive functioning and abstract reasoning (Brown et al., 2010; Green et al., 2009; Kully-Martens et al., 2013; Rasmussen et al., 2006; Schonfeld et al., 2006), attention (Connor et al., 1999; Green et al., 2009; Mattson et al., 2006), and adaptive behaviour and social skills (Greenbaum et al., 2009; Jirikowic et al., 2008; McGee et al., 2009; Nash et al., 2013; Rasmussen et al., 2010; Schonfeld et al., 2006; Streissguth et al., 2004).

Such individuals also have an empirically established increased risk for involvement in the criminal justice system (Fast et al., 1999; Schonfeld et al., 2005; Streissguth et al., 2004). Some researchers believe this connection to be a direct result of the neurocognitive deficits present in individuals with FASD (Brown et al., 2010; Fast et al., 1999; Manji et al., 2009) whereas others hypothesize the increased risk to be due to how the deficits present in social situations and thus leave the individual vulnerable to poor judgment and manipulation (Boland et al., 1998; Burd et al., 2010; Keil et al., 2010). Still others suggest that people with FASD are at a higher risk strictly because of the neglectful and abusive homes in which they are frequently raised, not the neurocognitive damage done in utero (Malone & Koren, 2012). Past research on FASD in offending populations is minimal and full of methodological problems, such as offender resistance and difficulty obtaining information on maternal consumption of alcohol. In addition, previous studies have focused almost exclusively on adult populations, with only one identified project looking at prenatal alcohol exposure in young offenders (Conry & Lane, 2009; as cited in Fast & Conry, 2009). The goal of the current project was to attempt to identify any neurocognitive or social differences between young offenders with and without a diagnosis of FASD, to help clarify where the difficulties lie for people with FASD.

In this sample, it was determined that young offenders with FASD were not qualitatively different from young offenders without FASD but were *more* impaired on neurocognitive measures across the board. This suggests that they are not dealing with different cognitive difficulties than other behaviourally challenged young offenders; however, they are likely more severely impaired and thus require additional assistance in managing said deficiencies. There was not found to be a direct relationship between any neurocognitive deficit and type or number of charges generated, which indicates that it is likely not the impairments themselves that cause the increased risk for criminal behaviour, but either the social environment in which such children are frequently raised or the interaction between the social environment and their neurocognitive impairment. The importance of home stability in this sample was clear, with youth coming from disruptive homes prior to the age of seven having accrued more charges than their peers in more stable homes; in addition, more young offenders with FASD had been placed in foster care than young offenders without a diagnosis. Substance use was not only more common in offenders with FASD but also more connected to their criminal behaviour, suggesting that the neurocognitive deficits may interact with social stimuli and situations (such as substance use) to produce an increase in risky behaviour.

This study was not able to identify the neurocognitive deficits most linked to crime in offenders with FASD, possibly because these deficits do not contribute to criminal activity. That is not a question that can be answered by this data set, as the comparison group and diagnosed group were both behaviourally challenged youth sent for assessment by the court; that differences in profiles were not found may be a function of the similarity of the groups being compared. However, what is clear is that youth with FASD are *more* neurocognitively impaired than their offending peers, even when compared to peers who are chronic offenders with behaviour problems. These impairments, viewed by professionals and society as “sick” when a child is young, become “bad” once the child is grown (Dej, 2011). This perception appears to be based on the notion that if appropriate services are rendered when the child is young enough, s/he should benefit from the interventions and the problems of FASD should become less pronounced as the child ages. Recent animal research offers hope that someday, postnatal interventions, such as environmental enrichment strategies, will be developed that can attenuate some of the negative cognitive and behavioural difficulties associated with FASD (Kelly et al., 2009b; Monk et al., 2012).

Yet to date, there is no indication in the literature that interventions designed to improve the neurocognitive damage done to the human brain in utero have been employed with any success (Denys et al., 2011). What is known in the literature on FASD in human populations is that the impairments appear to persist throughout the lifespan: “FASD are not childhood disorders that most people will simply outgrow but are lifelong disorders that change over the life of the affected individual” (Burd et al., 2010, p. 563). Buxton (2004) writes of how her daughter will need an “external forebrain” throughout her life span, to help her manage finances and tasks of daily living. Deficits in decision making in adolescents with FASD could not be distinguished from deficits in the youngest participants with FASD, suggesting the impairment persists at minimum into adolescence (Kully-Martens et al., 2013). In a review of the Coaching Families program run through Catholic Family Services, a program designed to assist families raising a child or children with FASD, families with an FASD child or youth identified the need for long-term support as one of the greatest challenges they face (Leenaars, Denys, Henneveld, & Rasussen, 2012). In fact, researchers note that it is likely that the role transitions from child to adolescent, and from adolescent to adult, are particularly vulnerable times for people with FASD, as the responsibilities placed on them increases while at the same time resources are more difficult for families to obtain (Dej, 2011; Denys et al., 2011; ICCFASD, 2011; Kully-Martens et al., 2013). In the same fashion, the transition from incarceration, with much structure and routine, to community living, where the governing rules are less clear and proscribed, can be particularly challenging for offenders with FASD (Burd et al., 2010).

It is important for a society to be consistent in how it approaches and handles mental and medical conditions. If deficits are thought to be representative of sickness in childhood, it should be questioned that these same deficits are viewed as representative as deviance or antisociality in older individuals dealing with increased pressures and reduced support. Dej (2011) refers to this as the “demedicalization” of FASD in adolescents and adults. As FASD is an invisible condition, often individuals with FASD are not given the same consideration for their behaviour as people with autism or Down’s syndrome, disabilities that are evident and easy to identify (Paley & Auerbach, 2010). If the central purpose of the criminal justice system is public safety, as the Correctional Service of Canada purports (Ogloff, 1995), it must be recognized that the current model of standard punishment has not been shown to be effective in reducing recidivism in such individuals. It is time to consider other options. Mela and Luther (2013) suggest a change in how

we view criminal responsibility to better reflect the spectrum of culpability as suggested by the neurocognitive literature, including a new plea of diminished responsibility for individuals with cognitive impairments. Alternative methods of behaviour modification (such as therapy and community-based programming) need to be considered before society concludes that people with FASD cannot or will not improve (ABA, 2012; CBA, 2010; Lafond, 2001). This statement is particularly true for young offenders, a population for which incarceration in general has proven to be a failure (Aiziz & Doyle, 2013).

7. REFERENCES

- Aase, J. M. (1994). Clinical recognition of FAS: difficulties of detection and diagnosis. *Alcohol Health & Research World*, 18(1), 5-9.
- Abel, E. L. (1998). *Fetal alcohol abuse syndrome*. New York: Plenum Press.
- Abkarian, G. G. (1992). Communication effects of prenatal alcohol exposure. *Journal of Communication Disorders*, 25, 221-240.
- Aegisdottir, S. (2007). The meta-analysis of clinical judgment project: Fifty-six years of accumulated research on clinical versus statistical prediction. *The Counseling Psychologist*, 34(3), 341-382.
- Aharoni E., Vincent, G. M., Harenski, C. L., Calhoun, V. D., Sinnott-Armstrong, W., Gazzaniga, M. S., et al. (2013). Neuroprediction of future arrest. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 100(15), 6223-6228. doi: 10.1073/pnas.1219302110
- Aizir, A., & Doyle, J. (July 16, 2013). What is the long-term impact of incarcerating juveniles? Retrieved June 15, 2013 from <http://www.voxeu.org/article/what-long-term-impact-incarcerating-juveniles>.
- Alberta Health Services. (2010). Estimating the rate of FASD in Canada. Women and Substance Use: Information Series. Retrieved Oct 25, 2011: 978-0-7785-8074-4 (web)
- Andrews, D. A., Bonta, J., & Wormith, J. S. (2011). The Risk-Need-Responsivity (RNR) model: Does adding the Good Lives model contribute to effective crime prevention? *Criminal Justice and Behaviour*, 38(7), 735-755.
- Archibald, S. L., Fennema-Notestine, C., Gamst, A., Riley, E. P., Mattson, S. N. and Jernigan, T. L. (2001), Brain dysmorphology in individuals with severe prenatal alcohol exposure. *Developmental Medicine & Child Neurology*, 43, 148–154. doi: 10.1111/j.1469-8749.2001.tb00179.x
- Archibald, S. J. & Kearns, K. A. (1999). Identification and description of new tests of executive functioning in children. *Child Neuropsychology*, 5(2), 115-129. doi: 10.1076/chin.5.2.115.3167
- Arendt, R. E., & Farkas, K. J. (2007). Maternal alcohol abuse and Fetal Alcohol Spectrum Disorder. *Alcoholism Treatment Quarterly*, 25(3), 3-20.
- Asato, M. R., Terwilliger, R., Woo, J., & Luna, B. (2010). White matter development in

- adolescence: A DTI study. *Cerebral Cortex*, 20, 2122-2131.
- Astley, S. J. (2006). Comparison of the 4-digit diagnostic code and the hoyme diagnostic guidelines for fetal alcohol spectrum disorders. *Pediatrics*, 118, 1532-1545.
- Astley, S. J. (2010). Profile of the first 1,400 patients receiving diagnostic evaluations for fetal alcohol spectrum disorder at the Washington state Fetal Alcohol Syndrome Diagnostic & Prevention Network. *Canadian Journal of Clinical Pharmacology*, 17(1), e132-e164.
- Astley, S. J., Aylward, E. H., Olson, H. C., Kerns, K., Brooks, A., Coggins, T. E., Davies, J., et al. (2009). Magnetic resonance imaging outcomes from a comprehensive magnetic resonance study of children with fetal alcohol spectrum disorders. *Alcoholism: Clinical and Experimental Research*, 33(10), 1671-1689.
- Astley, S. J. & Clarren, S. K. (2000). Diagnosing the full spectrum of fetal alcohol exposed individuals: Introducing the 4-Digit Diagnostic Code. *Alcohol & Alcoholism*, 35(4), 400-410.
- Astley, S. J., Stachowiak, J., Clarren, S. K., & Clausen, C. (2002). Application of the fetal alcohol syndrome facial photographic screening tool in a foster care population. *Journal of Pediatrics*, 141, 712-717.
- Autti-Ramo, I. (2000). Twelve-year follow-up of children exposed to alcohol in-utero. *Developmental Medicine and Child Neurology*, 42, 406-411.
- Baddeley, A. D. (1997). *Human Memory: Theory and Practice*. East Sussex: Psychology Press.
- Baer, J.S., Barr, H. M., Bookstein, F. L., Sampson, P. D., & Streissguth, A. P. (1998). Prenatal alcohol exposure and family history of alcoholism in the etiology of adolescent alcohol problems. *Journal of Studies on Alcohol and Drugs*, 59, 533-543.
- Baldo, J. V., Delis, D. C., Wilkins, D. P., & Shimamura, A. P. (2004). Is it bigger than a breadbox? Performance of patients with pre-frontal lesions on a new executive function test. *Archives of Clinical Neuropsychology*, 19, 407-419.
- Barab, S., & Squire, K. (2004). Design-based research: Putting a stake in the ground. *The Journal of the Learning Sciences*, 13(1), 1-14.
- Barnette, C. C. (1997). A judicial perspective on FAS: Memories of the making of Nanook of the North. In A. Streissguth & J. Kantor (Eds.), *The challenge of fetal alcohol syndrome: Overcoming secondary disabilities*, Seattle: University of Washington Press (pp. 134-145).

- Barth, R. P., Duncan, D. F., Hodorowicz, M. T., Kum, H. (2010). Felonious arrests of former foster care and TANF-involved youth. *Journal of the Society for Social Work and Research*, 1(2), 104-123. doi: 10.5243
- Baskin, D. R., & Sommers, I. (2011). Child maltreatment, placement strategies, and delinquency. *American Journal of Criminal Justice*, 36(2), 106-119. doi: 10.1007
- Bava, S., Frank, L. R., McQueeney, T., Schweinsburg, B. C., Schweinsburg, A. D., & Tapert, S. F. (2009). Altered white matter microstructure in adolescent substance users. *Psychiatry Research: Neuroimaging*, 173(3), 228-237. doi: <http://dx.doi.org/10.1016/j.psychresns.2009.04.005>
- Bava, S., Jacobus, J., Thayer, R. E., & Tapert, S. F. (2013). Longitudinal changes in white matter integrity among adolescent substance users. *Alcoholism: Clinical and Experimental Research*, 37(Suppl 1), E181-E189. doi: 10.1111/j.1530-0277.2012.01920.x
- Beatty, W. W., Jovic, Z., Monson, N., & Katzung, V. M. (1994). Problem solving by schizophrenic and schizoaffective patients on the Wisconsin and California card sorting tests. *Neuropsychology*, 8, 49-54.
- Beatty, W. W., & Monson, N. (1990). Problem solving in Parkinson's Disease: Comparison of performance on the Wisconsin and California Card Sorting Tests. *Journal of Geriatric Psychiatry and Neurology*, 3, 163-171. doi: 10.1177/089198879000300308
- Beets, M. W., & Foley, J. T. (2010). Comparison of 3 different analytic approaches for determining risk-related active and sedentary behaviour patterns in adolescents. *Journal of Physical Activity and Health*, 7, 381-392.
- Bell, I., & Mellor, D. (2009). Clinical judgments: Research and practice. *Australian Psychologist*, 44(2), 112-121.
- Belter, R. W., Duer, J. D., & Stanny, C. J. (1999). Clinical judgments regarding involuntary commitment: A comparison of mental health professions. *Journal of Psychological Practice*, 5(1), 32-38.
- Bennett, T., Holloway, K., & Fingleton, D. (2008). The statistical association between drug misuse and crime: A meta-analysis. *Aggression and Violent Behaviour*, 13(2), 107-118. doi: 10.1016/j.avb.2008.02.001
- Bergvall, A., Wessely, H., Forsman, A., & Hansen, S. (2001). A deficit in attentional set-shifting

- of violent offenders. *Psychological Medicine*, 31(6), 1095-1105. doi: 10.1017/S0033291701004317
- Berman, R. F., & Hannigan, J. H. (2000). Effects of prenatal alcohol exposure on the hippocampus: Spatial behaviour, electrophysiology, and neuroanatomy. *Hippocampus*, 10, 94-110.
- Best, D., Sidwell, C., Gossop, M., Harris, J., Strang, J. (2001). Crime and expenditure amongst polydrug misusers seeking treatment: The connection between prescribed methadone and crack use, and criminal involvement. *British Journal of Criminology*, 41(1), 119-126. doi: 10.1093/bjc/41.1.119
- Bisgard, E. B., Fisher, S., Adubato, S., Louis, M. (2010). Screening, diagnosis, and intervention with juvenile offenders. *Journal Psychiatry & Law*, 38, 475-506.
- Boland, F. J., Burrill, R., Duwyn, M., & Karp, J. (1998). *Fetal Alcohol Syndrome: Implications for Correctional Service* (Research report). Ottawa: Correctional Service of Canada.
- Boland, F. J., Chudley, A. E., & Grant, B. A. (2002). The challenge of Fetal Alcohol Syndrome in adult offender populations. *Forum of Correctional Research*, 14(3), 61-64.
- Bowes, N., Sutton, A., Jenkins, S., & McMurran, M. (2009). The alcohol treatment needs of violent and non-violent prisoners. *British Journal of Forensic Practice*, 11(1), 3-7.
- Brown, J. D., Riley, A. W., Walrath, C. M., Leaf, P. J., & Valdez, C. (2008). Academic achievement and school functioning among nonincarcerated youth involved with the juvenile justice system. *Journal of Education for Students Placed at Risk*, 13(1), 59-75.
- Brown, N. N., Wartnik, A. P., Connor, P. D., & Adler, R. S. (2010). A proposed model standard for forensic assessment of fetal alcohol spectrum disorders. *Journal of Psychiatry & Law*, 38, 383-418.
- Brown, T. L., Henggeler, S. W., Brondino, M. J., Pickrel, S. G. (1999). Trauma exposure, protective factors and mental health functioning of substance-abusing and dependent juvenile offenders. *Journal of Emotional and Behavioural Disorders*, 7(2), 94-102. doi: 10.1177/106342669900700204
- Bruskas, D. (2008). Children in foster care: A vulnerable population at risk. *Journal of Child and Adolescent Psychiatric Nursing*, 21(2), 70-77. doi: 10.1111/j.1744-6171.2008.00134.x
- Burd, L., Cotsonas-Hassler, T. M., Martsof, J. T., & Kerbeshian, J. (2003). Recognition and management of Fetal Alcohol Syndrome. *Neurotoxicology and Teratology*, 25, 681-688.

- Burd, L., Fast, D. K., Conry, J., & Williams, A. (2010). Fetal alcohol spectrum disorder as a marker for increased risk of involvement with correctional systems. *Journal of Psychiatry & Law*, 38, 559-583.
- Burd, L., Martsolf, J. T., Klug, M. G., & Kerbeshian, J. (2003). Diagnosis of FAS: A comparison of the Fetal Alcohol Syndrome Diagnostic Checklist and the Institute of Medicine Criteria for Fetal Alcohol Syndrome. *Neurotoxicology and Teratology*, 25, 719-724.
- Burgess, P. W., Alderman, N., Evans, J., Emslie, H., & Wilson, B. A. (1998). The ecological validity of tests of executive function. *Journal of the International Neuropsychological Society*, 4, 547-558.
- Buxton, B. (2004). *Damaged angels: A mother discovers the terrible cost of alcohol in pregnancy*. Toronto: Knopf Canada.
- Caldwell, R. M., Sturges, S. M., & Silver, N. C. (2007). Home versus school environments and their influences on the affective and behavioural states of African American, Hispanic, and Caucasian juvenile offenders. *Journal of Child and Family Studies*, 16, 125-138.
- Canada Newswire. (2009, October 21). Underfunded public services can't survive more cuts: OPSEU, p.
- Canadian Bar Association (CBA). (2010). *Fetal Alcohol Spectrum Disorder in the criminal justice system* (Resolution 10-02-A).
- Canadian Broadcasting Corporation (CBC; Sept. 14, 2007). Groups call for more services for adults with FASD. Retrieved June 5, 2013, from <http://www.cbc.ca/news/canada/manitoba/story/2007/09/14/fasd-adults.html>
- Caprara, D. L., Nash, K., Greenbaum, R., Rovet, J., & Koren, G. (2007). Novel approaches to the diagnosis of Fetal Alcohol Spectrum Disorder. *Neuroscience and Biobehavioural Reviews*, 31, 254-260.
- Carr, J. L., Agnihotri, S., & Keightley, M. (2010). Sensory processing and adaptive behaviour deficits of children across the fetal alcohol spectrum disorder continuum. *Alcoholism: Clinical and Experimental Research*, 34(6), 1022-1032.
- Carrion, V. G., & Steiner, H. (2000). Trauma and dissociation in delinquent adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(3), 353-359. doi: 10.1097/00004583-200003000-00018
- Center for Disease Control. (2002). Alcohol use among women of childbearing age –

- United States, 1991-1999. *Morbidity and Mortality Weekly Report*, 51, 273-276.
- Chand, A. (2000). The over-representation of Black children in the child protection system: Possible causes, consequences and solutions. *Child and Family Social Work*, 5, 67-77. doi: 10.1046/j.1365-2206.2000.00144.x
- Chasnoff, I. J., Wells, A. M., Telford, E., Schmidt, C., & Messer, G. (2010). Neurodevelopmental functioning in children with FAS, pFAS, and ARND. *Journal of Developmental and Behavioural Pediatrics*, 31, 192-201.
- Chen, J. (2012). Maternal alcohol use during pregnancy, birth weight, and early behavioural outcomes. *Alcohol and Alcoholism*, 47(6), 649-656.
- Chow, J. C., Jaffee, K., & Snowden, L. (2003). Racial/ethnic disparities in the use of mental health services in poverty areas. *American Journal of Public Health*, 93(5), 792-797.
- Chu, D. C. (2012). The links between religiosity, childhood sexual abuse, and subsequent marijuana use: An empirical inquiry of a sample of female college students. *International Journal of Offender Therapy and Comparative Criminology*, 56(6), 937-954. doi: 10.1177/0306624X11413560
- Chudley, A. E. (2009). Genetic predisposing factors in FASD. From IHE Consensus Development Conference, *Fetal Alcohol Spectrum Disorder (FASD) – Across the Lifespan*. Edmonton, AB.
- Chudley, A. E., Conry, J., Cook, J. L., Looock, C., Rosales, T., & LeBlanc, N. (2005). Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Canadian Medical Association Journal*, 172(5 suppl), s1-s21.
- Chudley, A. E., Kilgour, A. R., Cranston, M., & Edwards, M. (2007). Challenges of diagnosis in fetal alcohol syndrome and fetal alcohol spectrum disorder in the adult. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, 145C(3), 261-272. doi: 10.1002/ajmg.c.30140
- Church, M. W., & Kaltenbach, J. A. (1997). Hearing, speech, language, and vestibular disorders in the fetal alcohol syndrome: A literature review. *Alcoholism: Clinical and Experimental Research*, 21(3), 495-512. doi: 0145-6008/97/21035
- Clark, E., & Roman, D. D. (2006). Wisconsin Card Sorting Test. *Mental Measurements Yearbook and Test in Print*. Retrieved November 14, 2011 from <http://web.ebscohost.com.cyber.usask.ca/ehost/detail?vid=7&hid=126&sid=d4aef548->

- 2727-4c52-9856-8968086d1d16%40sessionmgr104&bdata=JnNpdGU9ZWhvc3QtbGl2ZQ%3d%3d#db=mmt&AN=TIP07002787
- Cohen, D. A., & Roman, D. D. (2006). Rey Complex Figure Test and Recognition Trial. *Mental Measurements Yearbook and Test in Print*. Retrieved November 9, 2011 from <http://web.ebscohost.com.cyber.usask.ca/ehost/detail?vid=6&hid=113&sid=1709a8d5-bc20-4938-a173-4220de2b0bb6%40sessionmgr112&bdata=JnNpdGU9ZWhvc3QtbGl2ZQ%3d%3d#db=mmt&AN=TIP07002167>.
- Cohen, J. (1988). *Statistical Power Analyses for the Behavioral Sciences* (2nd ed.). Hillsdale: Lawrence Erlbaum Associates, Publishers.
- Cohen, M. J. (1997). *Children's Memory Scale Manual*. San Antonio, TX: The Psychological Corporation.
- Coles, C. D., Goldstein, F. C., Lynch, M. E., Chen, X., Kable, J. A., Johnson, K. C., & Hu, X. (2011). Memory and brain volume in adults prenatally exposed to alcohol. *Brain and Cognition*, 75(1), 67-77.
- Coles, C. D., Lynch, M. E., Kable, J. A., Johnson, K. C., & Goldstein, F. C. (2010). Verbal and nonverbal memory in adults prenatally exposed to alcohol. *Alcoholism: Clinical and Experimental Research*, 34(5), 897-906.
- Comiskey, C. M., Stapleton, R., Kelly, P. A. (2012). Ongoing cocaine and benzodiazepine use: Effects on acquisitive crime committal rates amongst opiate users in treatment. *Drugs: Education, Prevention & Policy*, 19(5), 406-414. doi: 10.3109/09687637.2012.668977
- Connor, P. D., Streissguth, A. P., Sampson, P. D., Bookstein, F. L., & Barr, H. M. (1999). Individual differences in auditory and visual attention among fetal alcohol affected adults. *Alcoholism: Clinical and Experimental Research*, 23(8), 1395-1402.
- Conry, J. & Fast, D. K. (2000). Fetal alcohol syndrome and the criminal justice system. Vancouver: British Columbia Fetal Alcohol Syndrome Resource Society.
- Cowan, P. (2011, January 27). Sask. mental health system under strain. *Leader Post*. Retrieved February 18, 2011 from <http://www.leaderpost.com/health/Sask+mental+health+system+under+strain/4180908/story.html>

- Cox, L. V., Clairmont, D., Cox, S. (2008). Knowledge and attitudes of criminal justice professionals in relation to Fetal Alcohol Spectrum Disorder. *Canadian Journal of Clinical Pharmacology*, 15(2), e306-e313.
- Crocker, N., Vaurio, L., Riley, E. P., & Mattson, S. N. (2009). Comparison of adaptive behaviour in children with heavy prenatal alcohol exposure or Attention-Deficit/Hyperactivity Disorder. *Alcoholism: Clinical and Experimental Research*, 33(11), 2015-2023.
- Dagher-Margosian, J. (1997). Representing the FAS client in a criminal case. In: A. Streissguth & J. Kantor (Eds.), *The challenge of Fetal Alcohol Syndrome: Overcoming secondary disabilities* (p. 125-133). Seattle: University of Washington Press.
- D'Anguilli, A., Grunau, P., Maggi, S., & Herdman, A. (2006). Electroencephalographic correlates of prenatal exposure to alcohol in infants and children: A review of findings and implications for neurocognitive development. *Alcohol*, 40(2), 127-133. doi: 10.1016/j.alcohol.2006.09.031
- Darke, S., Torok, M., Kaye, S., Ross, J., McKetin, R. (2010). Comparative rates of violent crime among regular methamphetamine and opioid users: Offending and victimization. *Addiction*, 105(5), 916-919. doi: 10.1111/j.1360-0443.2009.02872.x
- Davis, A. (2010). *Handbook of Pediatric Neuropsychology*. New York: Springer Publishing.
- Dawes, R. M., Faust, D., & Meehl, P. E. (2002). Clinical versus actuarial judgment. In T. Gilovich, D. Griffin, & D. Kahneman (Eds.), *Heuristics and Biases: The Psychology of Human Judgment* (pp. 716-729). New York: Cambridge University Press.
- Dehaene, S., Molko, N., Cohen, L., & Wilson, A. J. (2004). Arithmetic and the brain. *Current Opinion in Neurobiology*, 14(2), 218-224. doi: 10.1016/j.conb.2004.03.008
- Dej, E. (2011). What once was sick is now bad: The shift from victim to deviant identity for those diagnosed with fetal alcohol spectrum disorder. *Canadian Journal of Society*, 36(2), 137-160.
- Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). *Delis-Kaplan Executive Function System (D-KEFS): Examiner's manual*. San Antonio, TX: The Psychological Corporation.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Holdnack, J. (2004). Reliability and validity of the Delis-Kaplan Executive Function System: An update. *Journal of the International Neuropsychological Society*, 10, 301-303. doi: 10.1017/S1355617704102191

- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. (2000). The California Verbal Learning Test—Second edition. San Antonio, TX: The Psychological Corporation.
- Denys, K., Rasmussen, C., Henneveld, D. (2011). The effectiveness of a community-based intervention for parents with FASD. *Community Mental Health Journal*, 47, 209-219. doi: 10.1007/s10597-009-9273-9
- Dicker, S., & Gordon, E. (2004). Building bridges for babies in foster care: The Babies Can't Quit initiative. *Juvenile and Family Court Journal*, 55(2), 29-41.
- Dishion, T. J., McCord, J., Poulin, F. (1999). When interventions harm: Peer groups and problem behaviour. *American Psychologist*, 54(9), 755-764. doi: 10.1037/0003-066X.54.9.755
- Disney, E. R., Iacono, W., McGue, M., Tully, E., & Legrand, L. (2008). Strengthening the case: Prenatal alcohol exposure is associated with increased risk for Conduct Disorder. *Pediatrics*, 122, e1225-e1230.
- Doig, J., McLennan, J. D., & Gibbard, W. B. (2008). Medication effects on symptoms of Attention-Deficit/Hyperactivity Disorder in children with Fetal Alcohol Spectrum Disorder. *Journal of Child and Adolescent Psychopharmacology*, 18(4), 365-371.
- Dolan, M., & Anderson, I. M. (2002). Executive and memory function and its relationship to trait impulsivity and aggression in personality disordered offenders. *Journal of Forensic Psychiatry*, 13(3), 503-526. doi: 10.1080/0958518021000019452
- Driver, D. (1999, November 23). Health-care funding disorganized, not underfunded. *Medical Post*, p. 42.
- Dubovsky, D. (2008). Co-occurring mental health disorders and misdiagnosis in FASD: Why do we need to know and what do we need to do about it. Presentation at National Fetal Alcohol Spectrum Disorders Conference: A Time for Change—Evidence and Strategies for Today, Tomorrow and Beyond. Madison, Wisconsin.
- Dunn, D. M., & Dunn, L. M. (2007). *Peabody picture vocabulary test: Manual*. San Antonio: Pearson Corporation.
- Eckstrand, K. L., Ding, Z., Dodge, N. C., Cowan, R. L., Jacobson, J. L., et al. (2012). Persistent dose-dependent changes in brain structure in young adults with low-to-moderate alcohol exposure in utero. *Alcoholism: Clinical and Experimental Research*, 36(11), 1892-1902. doi: 10.1111/j.1530-0277.2012.01819.x
- Ellis, L., & McDonald, J. N. (2001). Crime, delinquency, and social status: A reconsideration.

- Journal of Offender Rehabilitation*, 32(2), 23-52.
- Erickson, P. G., & Butters, J. E. (2005). How does the Canadian juvenile justice system respond to detained youth with substance use associated problems? Gaps, challenges, and emerging issues. *Substance Use and Misuse*, 40, 953-973.
- Farrington, D. P. (2011). Families and crime. In J. Q. Wilson & J. Petersilia (Eds.), *Crime and Public Policy* (pp. 130-157). New York: Oxford University press.
- Fast, D. K., & Conry, J. (2009). Fetal alcohol spectrum disorders and the criminal justice system. *Developmental Disabilities Research Review*, 15, 250-257.
- Fast, D. K., Conry, J., & Looock, C. A. (1999). Identifying fetal alcohol syndrome among youth in the criminal justice system. *Developmental and Behavioural Pediatrics*, 20(5), 370-372.
- Fazel, S., Bains, P., & Doll, H. (2006). Substance abuse and dependence in prisoners: A systematic review. *Addiction*, 101, 181-191.
- Feldman, H. S., Jones, K. L., Lindsay, S., Slymen, D., Klonoff-Cohen, H., Kao, K., et al. (2012). Prenatal alcohol exposure patterns and alcohol-related birth defects and growth deficiencies: A prospective study. *Alcoholism: Clinical and Experimental Research*, 36(4), 670-676.
- Fergusson, D. M., Horwood, L. J., Swain-Campbell, N. (2002). Cannabis use and psychosocial adjustment in adolescence and young adulthood. *Addiction*, 97(9), 1123-1135. doi: 10.1046/j.1360-0443.2002.00103.x
- Field, A. (2009). *Discovering Statistics Using SPSS, Third Edition*. Los Angeles: Sage Publications.
- Fife, C. (2006). Social workers in schools: The underfunded piece of the education puzzle. *Education Today*, 18(1), 24-26.
- Fisher, C. J. (2007). Validity of the Delis-Kaplan Executive Function System in pediatric populations. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 67(8-B), 4705.
- Franklin, L., Deitz, J., Jirikowic, T., & Astley, S. (2008). Children with fetal alcohol spectrum disorders: problem behaviours and sensory processing. *American Journal of Occupational Therapy*, 62(3), 265-273.
- Fraser, C. (2008). Victims and fetal alcohol spectrum disorders (FASD). *Victims of Crime*

Research Digest, 1, 22-28.

- Fraser, S. L., Muckle, G., Abdous, B. B., Jacobson, J. L., & Jacobson, S. W. (2012). Effects of binge drinking on infant growth and development in an Inuit sample. *Alcohol, 46*(3), 277-283.
- Frisell, T., Pawitan, Y., & Lanstrom, N. (2012). Is the association between general cognitive ability and violent crime caused by family-level confounders? *PLoS ONE, 7*(7). doi: e41783
- Fryer, S. L., McGee, C. L., Matt, G. E., Riley, E. P., & Mattson, S. N. (2007). Evaluation of psychopathological conditions in children with heavy prenatal alcohol exposure. *Pediatrics, 119*, e733-e741.
- Funder, D. C. (1995). On the accuracy of personality judgment: A realistic approach. *Psychology Review, 102*(4), 652-670.
- Furman, L., & Berman, B. W. (2004). Rethinking the AAP Attention Deficit/Hyperactivity Disorder guidelines. *Clinical Pediatrics, 43*, 601-603.
- Gagnon, M. P., Duplantie, J., Fortin, J. P., Landry, R. (2006). Implementing telehealth to support medical practice in rural/remote regions: What are the conditions for success? *Implementation Science, 1*(18). doi:10.1186/1748-5908-1-18.
- Ganellen, R. J. (2007). Assessing normal and abnormal personality functioning: Strengths and weakness of self-report, observer, performance-based methods. *Journal of Projective Techniques & Personality Assessment, 89*(1), 30-40.
- Gillespie, N. K., & Mckenzie, K. (2000). An examination of the role of neuropsychological deficits in mentally disordered sex offenders. *The Journal of Sexual Aggression, 5*, 21-29. doi: 10.1080/13552600008413293
- Gizzi, M. C., & Gerkin, P. (2010). Methamphetamine use and criminal behaviour. *International Journal of Offender Therapy and Comparative Criminology, 54*(6), 915-936. doi: 10.1177/0306624X09351825
- Goh, Y. I., Chudley, A. E., Clarren, S. K., Koren, G., Orrbine, E., Rosales, T., et al. (2008). Development of Canadian screening tools for fetal alcohol spectrum disorder. *Canadian Journal of Clinical Pharmacology, 15*(2), e344-e366.
- Goler, N. C., Armstrong, M. A., Osejo, V. M., Hung, Y., Haimowitz, M., & Caughey, A. B.

- (2012). Early Start: A cost-beneficial perinatal substance abuse program. *Obstetrics & Gynecology*, 119(1), 102-110. doi: 10.1097/AOG.0b013e31823d427d
- Goler, N. C., Armstrong, M. A., Taillac, C. J., Osejo, V. M. (2008). Substance abuse treatment linked with prenatal visits improves perinatal outcomes: A new standard. *Journal of Perinatology*, 28, 597-603.
- Goodman, L. A. (2002). Latent class analysis: The empirical study of latent types, latent variables, and latent structures. In J. A. Hagenaars & A. L. McCutcheon (Eds.), *Applied Latent Class Analysis* (p. 3-55). Cambridge: Cambridge University Press.
- Gray-Stanley, J. A. (2009). Stress and coping of direct care workers serving adults with intellectual/developmental disabilities. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 69(12-B), 7424.
- Green, C. R., Mihic, A. M., Nikkel, S. M., Stade, B. C., Rasmussen, C., Munoz, D. P., & Reynolds, J. N. (2009). Executive functioning in children with fetal alcohol spectrum disorders (FASD) measured using the Cambridge Neuropsychological Tests Automated Battery (CANTAB). *Journal of Child Psychology and Psychiatry*, 50(6), 688-697.
- Greenbaum, R. L., Stevens, S. A., Nash, K., Koren, G., Rovet, J. (2009). Social cognition and emotion processing abilities of children with fetal alcohol spectrum disorders: A comparison with Attention Deficit Hyperactivity Disorder. *Alcoholism: Clinical and Experimental Research*, 33(10), 1656-1670.
- Greenfield, R., & Valliant, P. M. (2007). Moral reasoning, executive function, and personality in violent and nonviolent adult offenders. *Psychological Reports*, 101, 323-333.
- Greve, K. W., Farrell, J. F., Besson, P. S., Crouch, J. A. (1995). A psychometric analysis of the California Card Sorting Test. *Archives of Clinical Neuropsychology*, 10(3), 265-278. doi: 0887-6177(94)00046-8
- Hancock, M., Tapscott, J. L., & Hoaken, P. N. S. (2010). Role of executive dysfunction in predicting frequency and severity of violence. *Aggressive Behaviour*, 36, 338-349.
- Hanlon, R. E., Rubin, L. H., Jensen, M., & Daoust, S. (2010). Neuropsychological features of indigent murder defendants and death row inmates in relation to homicidal aspects of their crimes. *Archives of Clinical Neuropsychology*, 25(1), 1-13. doi: 0.1093/arclin/acp099
- Hawkins, S. (2011). Education vs. incarceration. *American Prospect*, 22(1), A18-A19.

- Hays, D. G., Prosek, E. A., & McLeod, A. L. (2010). A mixed methodological analysis of the role of culture in the clinical decision-making process. *Journal of Counseling and Development*, 88(1), 114-121.
- Henry, J., Slaone, M., Black-Pond, C. (2007). Neurobiology and neurodevelopmental impact of childhood traumatic stress and prenatal alcohol exposure. *Language, Speech, and Hearing Services in Schools*, 38, 99-108. doi: 0161-1461/07/3802-0099
- Hepper, P. G., Dorman, J. C., Lynch, C. (2012). Fetal brain function in response to maternal alcohol consumption: Early evidence of damage. *Alcoholism: Clinical and Experimental Research*, 36(12), 2168-2175.
- Herrero, O., Escorial, S., & Colom, R. (2010). Basic executive processes in incarcerated offenders. *Personality and Individual Differences*, 48(2), 133-137.
doi.org/10.1016/j.paid.2009.09.009
- Hill, K., & Lightfoot, E. (2008). The dilemma between easing service access through a clear diagnosis of disability and unease in assigning labels to people with disabilities: A case study. *Journal of Social Work Values and Ethics*, 5(3). Retrieved from <http://www.socialworker.com/jswe/content/view/104/66/>
- Hongwanishkul, D., Happaney, K. R., Lee, W. S. C., & Zelazo, P. D. (2010). Assessment of hot and cool executive function in young children: Age-related changes and individual differences. *Developmental Neuropsychology*, 28(2), 617-644. doi 10.1207/s15326942dn2802_4
- Howell, K. K., Lynch, M. E., Platzman, K. A., Smith, G. H., & Coles, C. D. (2006). Prenatal alcohol exposure and ability, academic achievement, and school functioning in adolescence: A longitudinal follow-up. *Journal of Pediatric Psychology*, 31(1), 116-126.
- Hoyme, H. E., May, P. A., Kalberg, W. O., Koditwakku, P., Gossage, J. P., Trujillo, P. M., Buckley, D. G. et al. (2005). A practical clinical approach to diagnosis of fetal alcohol spectrum disorders: Clarification of the 1996 Institute of Medicine criteria. *Pediatrics*, 115, 39-47.
- Hsieh, C., & Pugh, M. D. (1993). Poverty, income inequality, and violent crime: A meta-analysis of recent aggregate data studies. *Criminal Justice Review*, 18(2), 182-202.
- Institute of Medicine. (1996). *Fetal Alcohol Syndrome: Diagnosis, epidemiology, prevention, and treatment*. Washington, D.C.: National Academy Press.

- Interagency Coordinating Committee on Fetal Alcohol Spectrum Disorders. (Oct. 31 – Nov. 2, 2011). Consensus statement on recognizing alcohol-related neurodevelopmental disorder (ARND) in primary health care of children. Rockville, MD.
- Jacobsen, S. E., Spregner, T., Andersson, S., Krogstad, J. (2003). Neuropsychological assessment and telemedicine: A preliminary study examining the reliability of neuropsychological services performed via telecommunications. *Journal of the International Neuropsychological Society*, 9(3), 472-478.
- Jacobson, S. W., Staton, M. E., Molteno, C. D., Burden, M. J., Fuller, D. S., Hoyme, H. E., Robinson, L. K., Khaole, N., & Jacobson, J. L. (2008). Impaired eyeblink conditioning in children with Fetal Alcohol Syndrome. *Alcoholism: Clinical and Experimental Research*, 32(2), 365-372.
- Jensen, P. S., Hoagwood, K., & Trickett, E. J. (1999). Ivory towers or earthen trenches? Community collaborations to foster real-world research. *Applied Developmental Science*, 3(4), 206-212.
- Jirikowic, T., Kartin, D., & Olson, H. C. (2008). Children with fetal alcohol spectrum disorders: A descriptive profile of adaptive function. *Canadian Journal of Occupational Therapy*, 75(4), 238-248.
- Johnson, M. K., Hashtroudi, S., & Lindsay, D. S. (1993). Source monitoring. *Psychological Bulletin*, 114(1), 3-28.
- Johnson, M. S. (2012). Resource allocation strategies and educational adequacy: Case studies of school level resource use in California middle schools. *Dissertation Abstracts International Section A: Humanities and Social Sciences*, 72(10-A), 3661.
- Jones, K. L., & Smith, D. W. (1973). Recognition of the fetal alcohol syndrome in early infancy. *Lancet*, 2, 999-1001.
- Jones, K. L., & Streissguth, A. P. (2010). Fetal alcohol syndrome and fetal alcohol spectrum disorders: A brief history. *Journal of Psychiatry & Law*, 38, 373-382.
- Joyal, C., Black, D. N., & Dassylva, B. (2007). The neuropsychology and neurology of sexual deviance: A review and pilot study. *Sexual Abuse: Journal of Research and Treatment*, 19(2), 155-173.
- Kalberg, W. O., Provost, B., Tollison, S. J., Tabachnick, B. G., Robinson, L. K., Hoyme, H. E.,

- Trujillo, P. M., Buckley, D., Aragon, A. S., & May, P. A. (2006). Comparison of motor delays in young children with fetal alcohol syndrome to those with prenatal alcohol exposure and with no prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, 30(12), 2037-2045.
- Katsiyannis, A., Ryan, J. B., Zhang, D., & Spann, A. (2008). Juvenile delinquency and recidivism: the impact of academic achievement. *Reading and Writing Quarterly*, 24(2), 177-196.
- Katsiyannis, A. & Archwamety, T. (1997). Factors related to recidivism among delinquent youths in a state correctional facility. *Journal of Child and Family Studies*, 6(1), 43-57.
- Keil, K. (July 14, 2011). Teen's rights not made clear: Judge. *Edmonton Journal*. Retrieved June 2nd from <http://www2.canada.com/edmontonjournal/news/story.html?id=fa1c8eb0-3640-4010-af02-37ec35f12ddb>.
- Keil, V., Paley, B., Frankel, F., & O'Connor, M. J. (2010). Impact of a social skills intervention on the hostile attributions of children with prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, 34(2), 231-241.
- Kelly, S. J., Goodlett, C. R., & Hannigan, J. H. (2009b). Animal models of fetal alcohol spectrum disorders: Impact of the social environment. *Developmental Disabilities Research Reviews*, 15, 200-208. doi: 10.1002/ddrr.69
- Kelly, T., Richardson, G., Hunter, R., & Knapp, M. (2002). Attention and executive function deficits in adolescent sex offenders. *Child Neuropsychology*, 8(2), 138-143.
- Kelly, Y. J., Sacker, A., Gray, R., Kelly, J., Wolke, D., Head, J., et al. (2010). Light drinking during pregnancy: Still no increased risk for socioemotional difficulties or cognitive deficits at 5 years of age? *Journal of Epidemiology and Community Health*, 66(1), 41-48.
- Kelly, Y. J., Sacker, A., Gray, R., Kelly, J., Wolke, D., Quigley, M. A. (2009a). Light drinking in pregnancy: A risk for behavioural problems and cognitive deficits at 3 years of age? *International Journal of Epidemiology*, 38, 129-140.
- Kerr, A., & Zelazo, P. D. (2004). Development of "hot" executive function: The children's gambling task. *Brain and Cognition*, 55(1), 148-157.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C., Hughes, M., Esleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Archives of General Psychiatry*, 51, 8-19.

- Khan, F., Turner-Stokes, L., Ng, L., & Kilpatrick, T. (2008). Multidisciplinary rehabilitation for adults with multiple sclerosis. *Journal of Neurology, Neurosurgery & Psychiatry*, 79(2), 114.
- Khan, S., Murray, R. P., & Barnes, G. E. (2002). A structural equation model of the effect of poverty and unemployment on alcohol abuse. *Addictive Behaviours*, 27(3), 405-423. doi: 10.1016/S0306-4603(01)00181-2
- Konrad, S. C. (2005). Mothers of children with acquired disabilities: Using the subjective voice to inform parent/professional partnership. *Omega: Journal of Death and Dying*, 51(1), 17-31.
- Kraanen, F. L., & Emmelkamp, P. M. (2011). Substance misuse and substance use disorders in sex offenders: a review. *Clinical Psychology Review*, 31(3), 478-489.
- Kully-Martens, K., Denys, K., Treit, S., Tamana, S., & Rasmussen, C. (2012a). A review of social skills deficits in individuals with fetal alcohol spectrum disorders and prenatal alcohol exposure: Profiles, mechanisms, and interventions. *Alcoholism: Clinical and Experimental Research*, 36(4), 568-576. 10.1111/j.1530-0277.2011.01661.x
- Kully-Martens, K., Pei, J., Job, J., & Rasmussen, C. (2012b). Source monitoring in children with and without fetal alcohol spectrum disorders. *Journal of Pediatric Psychology*, 37(7), 725-735. doi: 10.1093/jpepsy/jsr123
- Kully-Martens, K., Treit, S., Pei, J., & Rasmussen, C. (2012c). Affective decision-making on the Iowa Gambling Task in children and adolescents with fetal alcohol spectrum disorder. *Journal of the International Neuropsychological Society*, 19, 137-144. doi:10.1017/S1355617712001026
- LaDue, R. A., & Dunne, T. (1997). Legal issues and FAS. In A. P. Streissguth & J. Kanter (Eds.), *The Challenges of Fetal Alcohol Syndrome: Overcoming Secondary Disabilities*. Seattle: University of Washington Press.
- LaFond, M. E. (January 16, 2001). Her majesty the queen v. W. D.
- Lamberty, G. J., & Heilbronner, R. L. (2005). *The Clinical Practice of Neuropsychology: A survey of Practices and Settings*. Taylor & Francis e-Library. Retrieved November 1, 2011 from: <http://books.google.ca/books?id=9V1HGupPDMYC&printsec=copyright#v=onepage&q&f=false>.

- La Prairie, C. (2002). Aboriginal over-representation in the criminal justice system: A tale of nine cities. *Canadian Journal of Criminology*, 181, 181-208.
- Larkby, C. A., Goldschmidt, L., Hanusa, B. H., Day, N. L. (2011). Prenatal alcohol exposure is associated with conduct disorder in adolescence: Findings from a birth cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(3), 262-271.
- Latimer, J., & Foss, L. C. (2005). The sentencing of Aboriginal and non-Aboriginal youth under the young offenders act: A multivariate analysis. *Canadian Journal of Criminology and Criminal Justice*, 47(3), 481-500.
- Lebel, C., Rasmussen, C., Wyper, K., Andrew, G., & Beaulieu, C. (2010). Brain microstructure is related to math ability in children with Fetal Alcohol Spectrum Disorder. *Alcoholism: Clinical and Experimental Research*, 34(2), 354-363.
- Lebel, C., Rasmussen, C., Wyper, K., Walker, L., Andrew, G., Yager, J., Beaulieu, C. (2008). Brain diffusion abnormalities in children with fetal alcohol spectrum disorder. *Alcoholism: Clinical and Experimental Research*, 32(10), 1732-1740.
- Leenaars, L. S., Denys, K., Henneveld, D., & Rasmussen, C. (2012). The impact of fetal alcohol spectrum disorders on families: Evaluation of a family intervention program. *Community Mental Health Journal*, 48, 431-435. 10.1007/s10597-011-9425-6
- Lennings, C. J., Copeland, J., & Howard, J. (2003). Substance use patterns of young offenders and violent crime. *Aggressive Behaviour*, 29(5), 414-422. doi: 10.1002/ab.10048
- Levitt, S. D., & Lochner, L. (2001). The determinants of juvenile crime. In J. Gruber (Ed.), *Risky behaviour among youths: An economic analysis* (pp. 327-373).
- Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.
- Li, Y., & Lerner, R. M. (2011). Trajectories of school engagement during adolescence: Implications for grades, depression, delinquency, and substance use. *Developmental Psychology*, 47(1), 233-247.
- Lobmann, R., & Verthein, U. (2009). Explaining the effectiveness of heroin-assisted treatment on crime reductions. *Law and Human Behaviour*, 33(1), 83-95. doi: 10.1007/s10979-008-9138-8
- Loock, C. Conry, J., & Cook, J. L. (2005). Identifying Fetal Alcohol Spectrum Disorder in primary care. *Canadian Medical Association Journal*, 172(5), 628-630.

- Luna, B. (2009). Developmental changes in cognitive control through adolescence. *Advances in Child Development and Behaviour*, 37, 233-278. doi: 10.1016/S0065-2407(09)03706-9
- Lupton, C., Burd, L., & Harwood, R. (2004). Cost of Fetal Alcohol Spectrum Disorders. *American Journal of Medical Genetics Part C*, 127C, 42-50.
- Lynch, M. E., Coles, C. D., Corley, T., & Falek, A. (2003). Examining delinquency in adolescents differentially prenatally exposed to alcohol: The role of proximal and distal risk factors. *Journal of Studies on Alcohol*, 64(5), 678-686.
- MacCoun, R., Kilmer, B., & Reuter, P. (2003). Research on drugs-crime linkages: The next generation. *National Institute of Justice Special Report: Towards a Drugs and Crime Research Agenda for the 21st Century*, 65-95.
- MacLeod, S., & Koren, G. (2011). Meconium testing for fatty acid ethyl esters: A 2011 status report. *Journal of Population Therapeutics and Clinical Pharmacology*, 18(3), e500-e502.
- Malbin, D. V. (2004). Fetal alcohol spectrum disorder (FASD) and the role of family court judges in improving outcomes for children and families. *Juvenile and Family Court Journal*, 55(2), 53-63.
- Malone, M., & Koren, G. (2012). Alcohol-induced behavioural problems in fetal alcohol spectrum disorder versus confounding behavioural problems. *Journal of Popular Therapeutic Clinical Pharmacology*, 19(1), e32-e40.
- Manji, S., Pei, J., Loomes, C., & Rasmussen, C. (2009). A review of the verbal and visual memory impairments in children with foetal alcohol spectrum disorders. *Developmental Rehabilitation*, 12(4), 239-247.
- Manning, M. A., & Hoyme, H. E. (2007). Fetal alcohol spectrum disorders: A practical clinical approach to diagnosis. *Neuroscience and Biobehavioural Reviews*, 31, 230-238.
- Martinez, H. D. (2012). School level resource allocation to improve student performance: A case study of Orange County and Los Angeles County Title I elementary schools. *Dissertation Abstracts International Section A: Humanities and Social Sciences*, 72(10-A), 3664.
- Marzick, A. M. (2007). The Foster Care Ombudsman: Applying an international concept to help prevent institutional abuse of America's foster youth. *Family Court Review*, 45(3), 506-523. doi: 10.1111/j.1744-1617.2007.00166.x
- Mattson, S. N., Calarco, K. E., & Lang, A. R. (2006). Focused and shifting attention in children

- with heavy prenatal alcohol exposure. *Neuropsychology*, 20(3), 361-369.
- Mattson, S. N., Foroud, T., Sowell, E. R., Jones, K. L., Coles, C. D., Fagerlund, A. et al. (2010). Collaborative initiative on fetal alcohol spectrum disorders: Methodology of clinical projects. *Alcohol*, 44, 635-641.
- Mattson, S. N., Goodman, A. M., Caine, C., Delis, D. C., & Riley, E. P. (1999). Executive functioning in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, 23(11), 1808-1815.
- Mattson, S. N., & Riley, E. P. (1997). A review of the neurobehavioral deficits in children with fetal alcohol syndrome or prenatal exposure to alcohol. *Alcoholism: Clinical and Experimental Research*, 22, 279-294.
- Mattson, S. N., & Riley, E. P. (2000). Parent ratings of behavior in children with heavy prenatal alcohol exposure and IQ-matched controls. *Alcoholism: Clinical and Experimental Research*, 24(2), 226-231.
- Mattson, S. N., & Roebuck, T. M. (2002). Acquisition and retention of verbal and nonverbal information in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, 26(6), 875-882.
- Mattson, S. N., Roesch, S. C., Fagerlund, A., Autti-Ramo, I., Jones, K. L., May, P. A. et al. (2010a). Towards a neurobehavioural profile of fetal alcohol spectrum disorders. *Alcoholism: Clinical and Experimental Research*, 34(9), 1640-1650.
- May, P. A., Gossage, J. P., White-Country, M., Goodhart, K., DeCoteau, S., Trujillo, P. M., Kalberg, W. O. (2004). Alcohol consumption and other maternal risk factors for Fetal Alcohol Syndrome among three distinct samples of women before, during, and after pregnancy: The risk is relative. *American Journal of Medical Genetics, Seminars in Medical Genetics*, 127(C), 10-20.
- McConaughy, S. H. (2005). Direct observational assessment during test sessions and child clinical interviews. *School Psychology Review*, 34(4), 490-506.
- McGee, C. L., Bjorkquist, O. A., Price, J. M., Mattson, S. N., Riley, E. P. (2009). Social information processing skills in children with histories of heavy prenatal alcohol exposure. *Journal of Abnormal Child Psychology: An official publication of the International Society for Research in Child and Adolescent Psychopathology*, 37(6), 817-830.

- McGee, C. L., Bjorkquist, O. A., Riley, E. P., & Mattson, S. N. (2009). Impaired language performance in young children with heavy prenatal alcohol exposure. *Neurotoxicology and Teratology*, 31, 71-75.
- McGonnell, M., Corkum, P., McKinnon, M., MacPherson, M., Williams, T., Davidson, C., Jones, D. B., Stephenson, D. (2009). Doing it right: An interdisciplinary model for the diagnosis of ADHD. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, 18(4), 283-286.
- McIlroy, A. (May 27, 2011). Rewiring the brains of children with fetal alcohol syndrome. *The Globe and Mail*. Retrieved May 28, 2013, from <http://www.theglobeandmail.com/news/national/rewiring-the-brains-of-children-with-fetal-alcohol-syndrome/article1322351/>
- McMurran, M., Cusens, B. (2005). Alcohol and violent and non-violent acquisition offending. *Addiction Research and Theory*, 13(5), 439-443. doi: 10.1080/16066350500096058
- McMurty, R., & Curling, A. (2008). The review of the roots of youth violence, volume 2: Executive summary. Ontario: Queen's Printer.
- Meagher, D., O'Brien, S., Pulella, A., Oshun, A., & Brosnan, P. (2009). Multidisciplinary activities in a community mental health service: Relationship to Health of the Nation Outcome Scale scores and diagnosis. *Psychiatric Bulletin*, 33(5), 172-175.
- Mela, M., & Luther, G. (2013). Fetal alcohol spectrum disorder: Can diminished responsibility diminish criminal behaviour? *International Journal of Law and Psychiatry*, 36, 46-54.
- Meyer-Leu, Y., Sakari, L., Daeppen, J., Deriaz, O., & Gerber, S. (2011). Association of moderate alcohol use and binge drinking during pregnancy with neonatal health. *Alcoholism: Clinical and Experimental Research*, 35(9), 1669-1677.
- Meyers, J. E., & Meyers, K. (1996). *Rey complex figure test and recognition trials: Supplemental norms for children and adolescents*. Odessa, FL: Psychological Assessment Resources.
- Miller, T. R., Levy, D. T., Cohen, M. A., Cox, K. L. C. (2006). Costs of alcohol and drug-involved crime. *Prevention Science*, 7(4), 333-342. doi: 10.1007/s11121-006-0041-6
- Miura, H. (2009). Differences in frontal lobe function between violent and nonviolent conduct disorder in male adolescents. *Psychiatry and Clinical Neurosciences*, 63(2), 161-166. doi: 10.1111/j.1440-1819.2009.01935.x

- Mohr, J. J., Weiner, J. L., Chopp, R. M., & Wong, S. J. (2009). Effects of client bisexuality on clinical judgment: When is bias most likely to occur? *Journal of Counseling Psychology*, 56(1), 164-175.
- Monk, B. R., Leslie, F. M., & Thomas, J. D. (2012). The effects of perinatal choline supplementation on hippocampal cholinergic development in rats exposed to alcohol during the brain growth spurt. *Hippocampus*, 22(8), 1750-1757. doi: 10.1002/hipo.22009
- Moore, T. M., Stuart, G. L., Meehan, J. C., Rhatigan, D., Hellmuth, J. C., Keen, S. M. (2008). Drug abuse and aggression between intimate partners: A meta-analytic review. *Clinical Psychology Review*, 28(2), 247-274. doi: 10.1016/j.cpr.2007.05.003
- Moyer, M. W. (2013, January 4). How much alcohol is safe for expectant mothers? *Scientific American*. Retrieved May 28, 2013, from www.scientificamerican.com
- Murphy, K. R., & Davidshofer, C. O. (2005). *Psychological Testing: Principles and Applications* (6th ed.) Upper Saddle River, NJ: Pearson Education.
- Najavits, L. M., Runkel, R., Neuner, C., Frank, A. F., Thase, M. E., Crits-Christoph, P. et al. (2003). Rates and symptoms of PTSD among cocaine-dependent patients. *Journal of Studies on Alcohol*, 64(5), 601-606.
- Nanson, J. L., & Hiscock, M. (1990). Attention deficits in children exposed to alcohol prenatally. *Alcoholism: Clinical and Experimental Research*, 14(5), 656-661.
- Nardelli, A., Lebel, C., Rasmussen, C., Andrew, G., & Beaulieu, C. (2011). Extensive deep gray matter volume reductions in children and adolescents with fetal alcohol spectrum disorders. *Alcoholism: Clinical and Experimental Research*, 35(8), 1404-1417.
- Nash, K., Stevens, S., Rovet, J., Fantus, E., Nulman, I., Sorbara, D., et al. (2013). Towards identifying a characteristic neuropsychological profile for fetal alcohol spectrum disorders: 1. Analysis of the Motherisk FASD clinic. *Journal of Population Therapeutics and Clinical Pharmacology*, 20(1), e44-e52.
- Nelson, J. A. (2009). Rural communication technology. It's improving! *Home Health Care Management and Practice*, 21(6), 448-449.
- Nickerson, R. S. (2000). Null hypothesis significance testing: A review of old and continuing controversy. *Psychological Methods*, 5(2), 241-301.
- Nova, B., McDermott, B. E., Scott, C. L., Guillory, S. (2007). Sex offenders and insanity: An

- examination of 42 individuals found not guilty by reason of insanity. *Journal of the American Academy of Psychiatry and the Law*, 35, 444-450.
- O'Brien, W. H. (2010). Evaluating case formulation decision-making and therapist responsiveness: A perspective from the area of behavioral assessment and case formulation. *Pragmatic Case Studies in Psychology*, 6(4), 293-306.
- O'Brien, J. W., Norman, A. L., Fryer, S. L., Tapert, S. F., Paulus, M. P., Jones, K. L., et al. (2013). Effect of predictive cuing on response inhibition in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical & Experimental Research*, 37(4), 644-654. doi: 10.1111/acer.12017
- Odden, A. (1999). Case study 3: Formula funding of schools in the United States of America and Canada. In K. N. Ross & R. Levacic (Eds.), *Needs-Based Resource Allocation in Education via Formula Funding of Schools* (pp. 198-227).
- Ogloff, J. R. P. (1995). Information sharing and related ethical and legal issues for psychologists working in corrections. In T. A. Lies, L. L. Motiuk, & J. R. P. Ogloff (Eds.), *Forensic Psychology: Policy and Practice in Corrections*. Ottawa: Correctional Service of Canada.
- O'Leary, C. M., & Bower, C. (2012). Guidelines for pregnancy: What's an acceptable risk, and how is the evidence (finally) shaping up? *Drug and Alcohol Review*, 31(2), 170-183.
- Olson, H. C., Feldman, J. J., Streissguth, A. P., Sampson, P. D., & Bookstein, F. L. (1998). Neuropsychological deficits in adolescents with fetal alcohol syndrome: Clinical findings. *Alcoholism: Clinical and Experimental Research*, 22(9), 1998-2012.
- Paley, B., & Auerbach, B. E. (2010). Children with fetal alcohol spectrum disorders in the dependency court system: Challenges and recommendations. *Journal of Psychiatry & Law*, 38, 507-558.
- Parker, G. (2002). WRAT 3: Wide Range Achievement Test. *Test Review*, 5(1), 61-66.
- Parker, K. F., & Pruitt, M. V. (2000). Poverty, poverty concentration, and homicide. *Social Science Quarterly*, 81(2), 555-570.
- Parrish, J., Geary, E., Jones, J., & Seth, R. (2007). Executive functioning in childhood epilepsy: Parent-report and cognitive assessment. *Developmental Medicine and Child Neurology*, 49(6), 412-418.
- Peadon, E., Fremantle, E., Bower, C., & Elliott, E. J. (2008). International survey of diagnostic

- services for children with fetal alcohol spectrum disorders. *BMC Pediatrics*, 8(12). Retrieved June 15, 2011 from <http://www.biomedcentral.com/1471-2431/8/12>.
- Pei, J., Job, J., Kully-Martens, K., & Rasmussen, C. (2011). Executive function and memory in children with fetal alcohol spectrum disorder. *Child Neuropsychology*, 17(3), 290-309. doi: 10.1080/09297049.2010.544650
- Piquero, A. (2001). Testing Moffitt's neuropsychological variation hypothesis for the prediction of life-course persistent offending. *Psychology, Crime & Law*, 7(3), 193-215. doi: 10.1080/10683160108401794
- Plant, K. M., & Sanders, M. R. (2007). Predictors of care-giver stress in families of preschool-aged children with developmental disabilities. *Journal of Intellectual Disability Research*, 51(2), 109-124.
- Popova, S., Lange, S., Bekmuradov, D., Mihic, A., & Rehm, J. (2011). Fetal alcohol spectrum disorder prevalence estimates in correctional systems: A systematic literature review. *Canadian Journal of Public Health*, 102(5), 336-340.
- Popova, S., Stade, B., Lange, S., Rehm, J. (2012). A model for estimating the economic impact of fetal alcohol spectrum disorder. *Journal of Therapeutic Clinical Pharmacology*, 19(1), e51-e65.
- Pottick, K. J., Kirk, S. A., Hsieh, D. K., & Tian, X. (2007). Judging mental disorder in youths: Effects of client, clinician, and contextual differences. *Journal of Consulting and Clinical Psychology*, 75(1), 1-8.
- Raaijmakers, M. A. J., Smidts, D. P., Sergeant, J. A., Maassen, G. H., Posthumus, J. A., van Engeland, H., et al. (2008). Executive functions in preschool children with aggressive behaviour: Impairments in inhibitory control. *Journal of Abnormal Child Psychology*, 36, 1097-1107.
- Raine, A., Moffitt, T. E., Caspi, A., Loeber, R., Stouthamer-Loeber, M., & Lynam, D. (2005). Neurocognitive impairments in boys on the life-course persistent antisocial path. *Journal of Abnormal Psychology*, 114(1), 38-49.
- Rasmussen, C., Becker, M., McLennan, J., Urichuk, L., & Andrew, G. (2010). An evaluation of social skills in children with and without prenatal alcohol exposure. *Child: Care, Health, and Development*, 37(5), 711-718. doi:10.1111/j.1365-2214.2010.01152.x
- Rasmussen, C., & Bisanz, J. (2010). The relation between mathematics and working memory in

- young children with fetal alcohol spectrum disorders. *Journal of Special Education*, 45(3), 184-191. doi: 10.1177/0022466909356110
- Rasmussen, C., Horne, K., & Witol, A. (2006). Neurobehavioural functioning in children with fetal alcohol spectrum disorder. *Child Neuropsychology*, 12(6), 453-468.
- Rasmussen, C., Kully-Martens, K., Denys, K., Badry, D., Henneveld, D., Wyper, K., et al. (2012). The effectiveness of a community-based intervention program for women at-risk for giving birth to a child with fetal alcohol spectrum disorder (FASD). *Community Mental Health Journal*, 48, 12-21. doi: 10.1007/s10597-010-9342-0
- Rasmussen, C., Pei, J., Manji, S., Loomes, C., & Andrew, G. (2009). Memory strategy development in children with foetal alcohol spectrum disorders. *Developmental Neurohabilitation*, 12(4), 207-214. 10.1080/17518420902980126
- Razani, J., Casas, R., Wong, J. T., Lu, P., Alessi, C., & Josephson, K. (2007). Relationship between executive functioning and activities of daily living in patients with relatively mild dementia. *Applied Neuropsychology*, 14(3), 208-214. doi: 10.1080/09084280701509125
- Renn, P. (2000). The link between childhood trauma and later violent offending: A case study. In G. Boswell (Ed.), *Violent children and adolescents: Asking the question why* (pp. 71-90). London: Whurr.
- Ribeaud, D. (2004). Long-term impacts of the Swiss Heroin Prescription Trails on crime of treated heroin users. *Journal of Drug Issues*, 34(1), 163-194.
- Robinson, N. (February 25, 2013a). Jailing of kids 'does not work': WA governor Malcolm McCusker. *The Australian*. Retrieved May 29, 2013, from <http://www.theaustralian.com.au/national-affairs/indigenous/jailing-of-kids-does-not-work-governor/story-fn9hm1pm-1226584590355>
- Robinson, N. (February, 26, 2013b). Australia: Most needy kids at risk in sentencing push. *The Australian*. Retrieved May 29, 2013, from <http://www.theaustralian.com.au/national-affairs/indigenous/most-needy-kids-at-risk-in-sentencing-push/story-fn9hm1pm-1226585445246>
- Roebuck, T. M., Mattson, S. N., & Riley, E. P. (1999). Behavioural and psychosocial profiles of alcohol exposed children. *Alcoholism: Clinical and Experimental Research*, 23, 1070-1076.

- Romer, D. (2010). Adolescent risk taking, impulsivity, and brain development: Implications and prevention. *Developmental Psychobiology*, 52, 263-276. doi: 10.1002
- Rossow, I. (2004). Alcohol consumption and homicides in Canada, 1950-1999. *Contemporary Drug Problems: An Interdisciplinary Quarterly*, 31(3), 541-559.
- Royan, J., Tombaugh, T. N., Rees, L., & Francis, M. (2004). The Adjusting-Paced Serial Addition Test (Adjusting-PSAT): Thresholds for speed of information processing as a function of stimulus modality and problem complexity. *Archives of Clinical Neuropsychology*, 19, 131-143.
- Ruff, R. M., Light, R. H., & Parker, S. B. (1996). Benton controlled word association test: Reliability and updated norms. *Archives of Clinical Neuropsychology*, 11, 329-338.
- Ryan, J. P., Marshall, J. M., Herz, D., Hernandez, P. M. (2008). Juvenile delinquency in child welfare: Investigating group home effects. *Children and Youth Services Review*, 30(9), 1088-1099. doi: 10.1016
- Ryan, J. P., & Testa, M. F. (2005). Child maltreatment and juvenile delinquency: Investigating the role of placement and placement instability. *Children and Youth Services Review*, 27(3), 227-249. doi: 10.1016/j.childyouth.2004.05.007
- Sattler, J. M. (2008). *Assessment of Children: Cognitive Foundations, Fifth Edition*. San Diego: Sattler Publisher.
- Saltzman-Benaiah, J., Lalonde, C. E. (2007). Developing clinically suitable measures of social cognition for children: initial findings from a normative sample. *Clinical Neuropsychology*, 21, 294-317.
- Schonfeld, A. M., Mattson, S. N., Lang, A., Delis, D. C., & Riley, E. P. (2001). Verbal and nonverbal fluency in children with heavy prenatal alcohol exposure. *Journal of Studies on Alcohol*, 62, 239-246.
- Schonfeld, A. M., Mattson, S. N., Riley, E. P. (2005). Moral maturity and delinquency after prenatal alcohol exposure. *Journal of Studies on Alcohol and Drugs*, 66, 545-554.
- Schonfeld, A. M., Paley, B., Frankel, F., & O'Connor, M. J. (2006). Executive functioning predicts social skills following prenatal alcohol exposure. *Child Neuropsychology*, 12(6), 439-452.
- Schwartz, R. P., Jaffe, J. H., O'Grady, K. E., Kinlock, T. W., Gordon, M. S., Kelly, S. M. et al.

- (2009). Interim methadone treatment: Impact on arrests. *Drug and Alcohol Dependence*, 103(3), 148-154. doi: 10.1016/j.drugalcdep.2009.03.007
- Shelton, D. (2001). Emotional disorders in young offenders. *Journal of Nursing Scholarship*, 33(3), 259-263.
- Sheslow, D., & Adams, W. (2003). *Wide Range Assessment of Memory and Learning Second Edition: Administration and Technical Manual*. Wilmington, DE: Wide Range Inc.
- Silberg, J. L., Maes, H., Eaves, L. J. (2012). Unraveling the effect of genes and environment in the transmission of parental antisocial behaviour to children's conduct disturbance, depression, and hyperactivity. *Journal of Child Psychology and Psychiatry*, 53(6), 668-677.
- Silveri, M. M., Tzilos, G. K., & Yurgelun-Todd, D. A. (2008). Relationship between white matter volume and cognitive performance during adolescence: Effects of age, sex, and risk for drug use. *Addiction*, 103, 1509-1520. doi: 10.1111/j.1360-0443.2008.02272.x
- Simons, L., Ducette, J., Kirby, K. C., Stahler, G., Shipley, T. E. (2003). Childhood trauma, avoidance coping, and alcohol and other drug use among women in residential and outpatient treatment programs. *Alcoholism Treatment Quarterly*, 21(4), 37-54. doi: 10.1300/J020v21n04_04
- Sirin, S. R. (2005). Socioeconomic status and academic achievement: A meta-analytic review of research. *Review of Educational Research*, 75(3), 417-451.
- Smith, D. R. (2003). *Unveiling the WISC-IV*. The Psychological Corporation.
- Smyth, N. J., & Kost, K. A. (1998). Exploring the nature of the relationship between poverty and substance abuse: Knowns and unknowns. *Journal of Human Behaviour in the Social Environment*, 1(1), 67-82. doi: 10.1300/J137v01n01_05
- Sokol, R. J., Delaney-Black, V., & Nordstrom, B. (2003). Fetal Alcohol Spectrum Disorder. *Journal of the American Medical Association*, 290(22), 2996-2999.
- Song, Y., Skinner, J., Bynum, J., Sutherland, J., Wennberg, J. E., & Fisher, E. S. (2010). Regional variations in diagnostic practices. *The New England Journal of Medicine*, 363(1), 45-53.
- Sowell, E. R., Mattson, S. N., Kan, E., Thompson, P. M., Riley, E. P., Toga, A. W. (2008).

- Abnormal cortical thickness and brain-behaviour correlation patterns in individuals with heavy prenatal alcohol exposure. *Cerebral Cortex*, 18, 136-144. doi: 10.1093/cercor/bhm039
- Spano, R. (2005). Potential sources of observer bias in police observational data. *Social Science Research*, 34(3), 591-617.
- Spunt, B., Brownstein, H., Goldstein, P., Fendrich, M., & Liberty, H. J. (1995). Drug use by homicide offenders. *Journal of Psychoactive Drugs*, 27(2), 125-134. doi: 10.1080/02791072.1995.10471681
- Steinhausen, H. C., & Spohr, H. L. (1998). Long-term outcome of children with Fetal Alcohol Syndrome: Psychopathology, behaviour, and intelligence. *Alcoholism: Clinical and Experimental Research*, 22(2), 334-338.
- Steinhausen, H. C., Willms, J., Metzke, C. W., & Spohr, H. L. (2003). Behavioural phenotype in Foetal Alcohol Syndrome and Foetal Alcohol Effects. *Developmental Medicine & Child Neurology*, 45, 179-182.
- Steinhausen, H. C., Willms, J., & Spohr, H. L. (1993). Long-term psychopathological and cognitive outcome of children with fetal alcohol syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 990-994.
- Streissguth, A. P., Aase, J. M., Clarren, S. K., Randels, S. P., LaDue, R. A., & Smith, D. F. (1991). Fetal alcohol syndrome in adolescents and adults. *Journal of the American Medical Association*, 265(15), 1961-1967.
- Streissguth, A., Barr, H., Kogan, J., & Bookstein, F. (1997). Primary and secondary disabilities in Fetal Alcohol Syndrome. In: A. Streissguth, J. Kantor (Eds). *The challenge of Fetal Alcohol Syndrome: Overcoming secondary disabilities* (p. 25-39). Seattle: University of Washington Press.
- Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'Malley, K., & Young, J. K. (2004). Risk factors for adverse life outcomes in Fetal Alcohol Syndrome and Fetal Alcohol Effects. *Developmental and Behavioural Pediatrics*, 25(4), 228-238.
- Streissguth, A. P., & O'Malley, K. (2000). Neuropsychiatric implications and long-term consequences of fetal alcohol spectrum disorders. *Seminars in Clinical Neuropsychiatry*, 5(3), 177-190.
- Stephen, J. M., Kodituwakku, P. W., Kodituwakku, E. L., Romero, L., Peters, A. M.,

- Sharadamma, N. M., et al. (2012). Delays in auditory processing identified in preschool children with FASD. *Alcoholism: Clinical and Experimental Research*, 36(10), 1720-1727. doi: 10.1111/j.1530-0277.2012.01769.x
- Stettner, G. M., Kubin, L., Volgin, D. V. (2011). Antagonism of orexin 1 receptors eliminates motor hyperactivity and improved homing response acquisition in juvenile rats exposed to alcohol during early postnatal period. *Behavioural Brain Research*, 221(1), 324-328. doi: 10.1016/j.bbr.2011.03.028
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms and commentary* (3rd ed.). New York: Oxford University Press.
- Strong, C. H., Tiesma, D., & Donders, J. (2011). Criterion validity of the Delis-Kaplan Executive Function System (D-KEFS) fluency subtests after traumatic brain injury. *Journal of the International Neuropsychological Society*, 17, 230-237. doi: 10.1017/S1355617710001451
- Substance Abuse and Mental Health Service Administration, US Department of Health and Human Services. (2006). *How fetal alcohol spectrum disorders co-occur with mental illness*. Retrieved Oct 23, 2010 from: fasdcenter.samhsa.gov/documents/WYNK_CoOccurMentalIllnes.pdf.
- Sullivan, C. J. (2006). Early adolescent delinquency: Assessing the role of childhood problems, family environment, and peer pressure. *Youth Violence and Juvenile Justice*, 4, 291-314.
- Tabachnick, B. G., & Fidell, L. S. (2006). *Using multivariate statistics* (5th ed.). Boston: Pearson/Allyn & Bacon.
- Thalheimer, W., & Cook, S. (2002, August). *How to calculate effect sizes from published research articles: A simplified methodology*. Retrieved April 1, 2014 from http://work-learning.com/effect_sizes.htm.
- Tardiff, K., Marzuk, P. M., Leon, A. C., Hirsch, C. S., Stajic, M., Portera, L., et al. (1994). Homicide in New York City: Cocaine use and firearms. *The Journal of the American Medical Association*, 272(1), 43-36. doi: 10.1001/jama.272.1.43
- Temple, J. R., Shorey, R. C., Fite, P., Stuart, G. L., & Le, V. D. (2013). Substance use as a longitudinal predictor of the perpetration of teen dating violence. *Journal of Youth and Adolescence*, 42, 596-606. doi: 10.1007/s10964.012.9877.1
- Tiihonen, J., Eronen, M., & Hakola, P. (1993). Criminality associated with mental disorders and

- intellectual deficiency. *Archives of General Psychiatry*, 50(11), 917-918.
- Timko, C., Moos, B. S., Moos, R. H. (2009). Gender differences in 16-year trends in assault- and police-related problems due to drinking. *Addictive Behaviours*, 34(9), 744-750. doi: 10.1016/j.addbeh.2009.04.013
- Trocmé, N., Knoke, D., & Blackstock, C. (2004). Pathways to the overrepresentation of Aboriginal children in Canada's child welfare system. *Social Service Review*, 78(4), 577-600.
- Tsai, S., & Wang, H. (2009). The relationship between caregiver's strain and social support among mothers with intellectually disabled children. *Journal of Clinical Nursing*, 18(4), 539-548.
- Tulsky, D., & Zhu, J. (1997). *WAIS-III/WMS-III Technical Manual*. San Antonio: Psychological Corporation.
- van der Laan, A., Veenstra, R., Bogaerts, S., Verhulst, F. C., & Ormel, J. (2010). Serious, minor, and non-delinquents in early adolescence: The impact of cumulative risk and promotive factors. The TRAILS study. *Journal of Abnormal Child Psychology*, 38, 339-351.
- Vaurio, L., Riley, E. P., & Mattson, S. N. (2008). Differences in executive functioning in children with heavy prenatal alcohol exposure or attention-deficit/hyperactivity disorder. *Journal of the International Neuropsychological Society*, 14, 119-129.
- Vermunt, J. K., & Magidson, J. (2002). Latent class cluster analysis. In J. A. Hagenaars & A. L. McCutcheon (Eds.), *Applied Latent Class Analysis* (p. 89-106). Cambridge: Cambridge University Press.
- Waisbren, S. E., Hanley, W., Levy, H. L., Shifrin, H., Allred, E., Azen, C., Chang, P.N. et al. (2000). Outcome at 4 years in offspring of woman with maternal phenylketonuria: The Maternal PKU Collaborative Study. *Journal of the American Medical Association*, 283, 756-762.
- Wechsler, D. (2003). *Wechsler Intelligence Scale for Children – Fourth Edition: Technical and interpretive manual*. San Antonio: The Psychological Corporation.
- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV)*. San Antonio, TX: Pearson.
- Wedding, D., Kohout, J., Mengel, M. B., Ohlemiller, M., Ulione, M., Cook, K., Rudeen, K. et al.

- (2007). Psychologists' knowledge and attitudes about Fetal Alcohol Syndrome, fetal alcohol spectrum disorders, and alcohol use during pregnancy. *Professional Psychology: Research and Practice*, 38(2), 208-213.
- White, P., Edwards, N., & Townsend-White, C. (2006). Stress and burnout amongst professional carers of people with intellectual disability: Another health inequity. *Current Opinion in Psychiatry*, 19(5), 502-507.
- Wikstrom, P. H. (1991). Housing tenure, social class, and offending: The individual-level relationship in child and youth. *Criminal Behaviour and Mental Health*, 1(1), 69-89.
- Wilkinson, G. S., & Robertson, G. J. (2006). *Wide Range Achievement Test®—Fourth Edition (WRAT4®)*. Lutz, FL: Psychological Assessment Resources, Inc.
- Williams, K. T. (2007). *EVT-2: Expressive Vocabulary Test*. Minneapolis, MN: Pearson Assessments.
- Wilson, D., & Macdonald, D. (2010). *The income gap between Aboriginal peoples and the rest of Canada*. Ottawa: Canadian Centre for Policy Alternatives. Retrieved from <http://mail.policyalternatives.org/sites/default/files/uploads/publications/reports/docs/Aboriginal%20Income%20Gap.pdf>
- Winefield, H. R., Barlow, J., & Harvey, E. (1998). Responses to support groups for family caregivers in schizophrenia: Who benefits from what? *Australian and New Zealand Journal of Mental Health Nursing*, 7(3), 103-110.
- Wozniak, J. R., Mueller, B. A., Chang, P. N., Muetzel, R. L., Caros, L., & Lim, K. O. (2006). Diffusion tensor imaging in children with fetal alcohol spectrum disorders. *Alcoholism: Clinical and Experimental Research*, 30, 1799–1806.
- Wurst, F. M., Alexson, S., Wolferdorf, M., Bechtel, G., Forster, S., Alling, C., & Aradottir, S. (2004). Concentration of fatty acid ethyl esters in hair of alcoholics: Comparison to other biological state markers and self-reported ethanol intake. *Alcohol and Alcoholism*, 39, 33-38.
- Yang, Y., Rousotte, F., Kan, E., Sulik, K. K., Mattson, S. N., Riley, E. P., et al. (2011). Abnormal cortical thickness alterations in fetal alcohol spectrum disorders and their relationships with facial dysmorphology. *Cerebral Cortex*, 22, 1170-1179.
- Yessine, A. K., & Bonta, J. (2009). The offending trajectories of youthful Aboriginal offenders. *Canadian Journal of Criminology and Criminal Justice*, 51(4), 435-472.

- Zabel, R. H., & Nigro, F. A. (2001). The influence of special education experience and gender of juvenile offenders on academic achievement scores in reading, language, and mathematics. *Behavioural Disorders*, 26(2), 164-172.
- Zagar, R. J., Busch, K. G., Grover, W. M., Hughes, J. R., & Arbit, J. (2009). Looking forward and backward in records for risks among homicidal youth. *Psychological Reports*, 104, 103-127.
- Zelner, I., Shor., S., Lynn, H., Roukema, H., Lum, L., Elsinga, K., et al. (2012). Clinical use of mocnium fatty acid ethyl esters for identifying children at risk for alcohol-related disabilities: The first reported case. *Journal of Population Therapeutics and Clinical Pharmacology*, 19(1), e26-e31.
- Zhou, D., Lebel, C., Lepage, C., Rasmussen, C., Evans, A., Wyper, K., et al. (2011). Developmental cortical thinning in fetal alcohol spectrum disorders. *NeruoImage*, 58, 16-25. doi: 10.1016/j.neuroimage.2011.06.026